# USMLE
# STEP 2 C
## nical Knowled

Ninth Edition

**A STUDENT-TO-STUDENT GUIDE**

hensive high-yield review for the USMLE St

# USMLE Step 2 C

## Ninth Edition

TAO LE, MD, MHS
Associate Clinical Professor
Chief, Section of Allergy and Immunology
Department of Medicine
University of Louisville

VIKAS BHUSHAN, MD
Diagnostic Radiologist
Los Angeles

VINCENT L. CHEN, MD
Resident, Department of Internal Medicine
Stanford University Medical Center

MICHAEL R. KING, MD

able at special quantity discounts to use as premiums and sales promotions or for use in corporate trai
mhprofessional.com.

2001 by The McGraw-Hill Companies, Inc; copyright © 1999, 1996 by Appleton and Lange.

rk of McGraw-Hill Education.

**NOTICE**

new research and clinical experience broaden our knowledge, changes in treatment and drug therapy a
believed to be reliable in their efforts to provide information that is complete and generally in accor
possibility of human error or changes in medical sciences, neither the authors nor the publisher nor a
k warrants that the information contained herein is in every respect accurate or complete, and they di
n use of the information contained in this work. Readers are encouraged to confirm the information
vised to check the product information sheet included in the package of each drug they plan to adm
at changes have not been made in the recommended dose or in the contraindications for administrati
requently used drugs.

-Hill Education and its licensors reserve all rights in and to the work. Use of this work is subject t
o store and retrieve one copy of the work, you may not decompile, disassemble, reverse engineer, re
ate, sell, publish or sublicense the work or any part of it without McGraw-Hill Education's prior con
er use of the work is strictly prohibited. Your right to use the work may be terminated if you fail to c

McGRAW-HILL EDUCATION AND ITS LICENSORS MAKE NO GUARANTEES OR WA
F OR RESULTS TO BE OBTAINED FROM USING THE WORK, INCLUDING ANY INFOR
INK OR OTHERWISE, AND EXPRESSLY DISCLAIM ANY WARRANTY, EXPRESS OR IMPL
CHANTABILITY OR FITNESS FOR A PARTICULAR PURPOSE. McGraw-Hill Education and
will meet your requirements or that its operation will be uninterrupted or error free. Neither McGraw
uracy, error or omission, regardless of cause, in the work or for any damages resulting therefrom. McG
ed through the work. Under no circumstances shall McGraw-Hill Education and/or its licensors be l
s that result from the use of or inability to use the work, even if any of them has been advised of the po
use whatsoever whether such claim or cause arises in contract, tort or otherwise.

*This page intentionally left blank*

2 Defining Your Goal

When To Take the Exam

mputer-Based Testing Basics 2

TRUc TUREd? 2

AT WILL THE c BT BE LIKE? 3

RMAT MEAN Fo R ME? 3

AKE THE EXAMINATIo N? 4

lEd ULE THE EXAMINATIo N? 5

5

6

AMINATIo N, WHAT HAPPENS To MY Sc o RE? 6

NS ARE ASKEd? 6

To WAIT BEFo RE I GET MY Sc o RES? 7

Po RTEd? 7

Study Resources

QUALITY c o NSId ERATIo NS

c LINIc AL REVIEW Bo o KS

TEST BANKS

TEXTS ANd No TES

c o MMERc IAL c o URSES

NBME/USMLE PUBLIc ATIo NS

Test-Day Checklist

THINGS To BRING WITH Yo U To THE Ex

Testing Agencies

- The Step 2 Clinical Knowledge examination (Step 2 C
- The Step 2 Clinical Skills examination (Step 2 CS)

The USMLE Step 2 CK is the second of three examina
pass in order to become a licensed physician in the Unit
puterized Step 2 CK is a 1-day (9-hour) multiple-choice e

Students are also required to take the Step 2 CS, a 1-day
which students examine 12 standardized patients. For m
this examination, please refer to First Aid for the USMLE
tion about the Step 2 CS format and about eligibility, regi
can be found at http://www.nbme.org.

The information found in this section as well as in the re
will address only the Step 2 CK.

## USMLE Step 2 CK—Computer-Based T

### HOW WILL THE CBT BE STRUCTURED?

The Step 2 CK is a computer-based test (CBT) adminis
Inc. It is a 1-day examination with approximately 352 qu
eight 60-minute blocks of 44 questions each, administere
testing session. Some sections may have more or fewer th
the maximum question total for the exam is 355. The Step
**FRED v2** software program as that used on the USMLE St

Three question styles predominate throughout the exa
common format is **Single One Best Answer** questions. T
multiple-choice format in which you are tasked with
correct" answer. Another common style is **Matching Se**
consist of a series of questions related to a similar topic

w.usmle.org) and try out prior to the examination. If you with the FRED v2 testing interface ahead of time, you nute tutorial offered on examination day and add those tted break time of 45 minutes.

, examinees are not allowed to bring personal equipment ed for medical reasons and soft-foam earplugs as detailed sting area—which means that writing implements, out- ven analog), cellular telephones, and electronic paging hibited. Food and beverages are prohibited as well. The ou a small locker in which you can store your belongings ing for the day. Examinees will also be given 2 (8″ × 11″) rfaces, pens, and erasers for note taking and for recording Identification Number (CIN). These materials must be xamination. Testing centers are monitored by audio and quipment. Each time you enter the testing room, you will screening process to ensure that you are not bringing in

familiar with a typical question screen. A window to the questions in the block and shows you the unanswered with an "i"). Some questions will contain figures, color or video adjacent to the question. Although the con- of the screen can be adjusted, there are no other ways to ure (eg, zooming or panning). Larger images are accessed utton. The examinee can also call up a window displaying You may **mark** questions to review at a later time by click- at the top of the screen. The **annotation** feature functions y erase sheets and allows you to jot down notes during the with the **highlighting/strike-out** and annotation features nd multiple answers.

a few practice blocks to determine which tools actually

Keyboar

- A–E
- Ente next

for students to assess their Step 2 CK knowledge with
Clinical Science Self-Assessment (CCSSA) test. This tes
NBME Web site in several versions for $50, or $60 for
which will display at the end of the exam all of the que
incorrectly, without additional explanations. The cont
items resembles that of the USMLE Step 2 CK. Upon
CCSSA, users will be provided with a performance pro
strengths and weaknesses. This feedback is intended for
only and is not necessarily an indicator of Step 2 CK per
information on the CCSSA examination, visit the NBME
www.nbme.org, and click on the link for "NBME Web-ba
Service."

## HOW DO I REGISTER TO TAKE THE EXAMINATION?

Information on Step 2 CK format, content, and registratio
be found on the USMLE Web site. To register for the exa
graduates of accredited schools in the United States and
online at the NBME Web site (http://www.nbme.org).
graduates of non-US/Canadian schools should apply throu
Commission for Foreign Medical Graduates ECFMG
.org). A printable version of the application is also avail
The preliminary registration process for the USMLE Step

- Complete a registration form, and send your exam
  NBME (online).
- Select a 3-month block in which you wish to be te
  August).
- Attach a passport-type photo to your completed applica
- Complete a Certification of Identification and Autho
  form must be signed by an official at your medical sch
  istrar's office (if you are a student) or a notary public
  ated) to verify your identity. It is valid for 5 years, allow

ccess to the codes and will not be able to supply these t **lose your permit!** You will not be allowed to take the ou present your permit along with an unexpired, govern- identification that contains your signature (eg, driver's Iake sure the name on your photo ID exactly matches the n your scheduling permit.

## RESCHEDULE THE EXAMINATION?

ur date and/or center within your 3-month period by con- space is available. If you reschedule 31 days before your te, there is no fee; between 6 and 30 days before, there is a days before, there is a larger fee. If you need to reschedule 3-month period, you can apply for a single 3-month exten- /June can be extended through July/August/September) period has begun (visit http://www.nbme.org for more extension currently costs $70. For other rescheduling mit a new application along with another application fee.

## ?

nterest on the CBT examination. Here is a breakdown of edule:

| | |
|---|---|
| | 15 minutes |
| cks (44 questions per block) | 8 hours |
| ne for lunch) | 45 minutes |
| | 9 hours |

Because
is sched
served"
Prometr
schedul

## IF I LEAVE DURING THE EXAMINATION, WHAT HAPPENS I

You are considered to have started the examination onc
your CIN onto the computer screen. In order to receiv
however, you must finish the entire examination. This m
start and either finish or run out of time for each block of
you do not complete all the question blocks, your examin
mented on your USMLE score transcript as an incompl
actual score will be reported.

The examination ends when all blocks have been com
expired. As you leave the testing center, you will receive a
tion notice to document your completion of the examinat

## WHAT TYPES OF QUESTIONS ARE ASKED?

The Step 2 CK is an integrated examination that tests unde
conditions, disease categories, and physician tasks. Almost
examination are case based. A substantial amount of extr
may be given, or a clinical scenario may be followed by a
be answered without actually requiring that you read the c
determine which information is superfluous and which is
at hand. Content areas include internal medicine, OB/G
ventive services, psychiatry, surgery, and other areas relevan
care under supervision. Physician tasks are distributed as fo

- Establishing a diagnosis (25–40%)
- Understanding the mechanisms of disease (20–35%)
- Applying principles of management (15–25%)
- Promoting preventive medicine and health maintenan

Most questions on the examination have a **Single Best**
some **Matching Sets** and **Sequential Item Sets** will be fo

the Step 2 CK is that no matter how much you study, estions you will not be able to answer with confidence. If question cannot be solved in a reasonable period of time, guess and move on; you will not be penalized for guess- ind that some of the USMLE examination questions are will not count toward your score.

## HAVE TO WAIT BEFORE I GET MY SCORES?

ts scores 3–4 weeks after the examinee's test date. Dur- owever, as many as 6 weeks may pass before reports are rmation concerning the time required for score reporting MLE Web site, http://www.usmle.org.

## RES REPORTED?

e report, your Step 2 CK report includes your pass/fail sta- e, and a performance profile organized by discipline and Figures 1-1A and 1-1B). The score is a 3-digit scaled score ed proficiency standard. As of July 1, 2014, the required . This score requires answering 60–70% of questions cor- ents in the required passing score will be available on the

## Your Goa

important thing to do in your Step 2 CK preparation is want to do on the exam, as this will ultimately determine ation that will be necessary. The amount of time spent in examination varies widely among medical students. Pos- he following:

The USMLE is a single examination program consisting of three Steps designed to understanding of and ability to apply concepts and principles that are important in hea constitute the basis of safe and effective patient care. Step 2 is designed to assess whether medical knowledge, skills, and understanding of clinical science essential for the provisio supervision, including emphasis on health promotion and disease prevention. The inclusion sequence is intended to ensure that due attention is devoted to principles of clini patient-centered skills that provide the foundation for the safe and competent practice of m components to Step 2: a Clinical Knowledge (CK) examination and a Clinical Skills (CS) e represents results for the Step 2 CK examination only. Results of the examination are repo authorities in the United States and its territories for use in granting an initial license to pract represents your result for the administration of Step 2 CK on the test date shown above.

| | |
|---|---|
| PASS | This result is based on the minimum passing score recommended by Individual licensing authorities may accept the USMLE-recommend establish a different passing score for their own jurisdictions. |

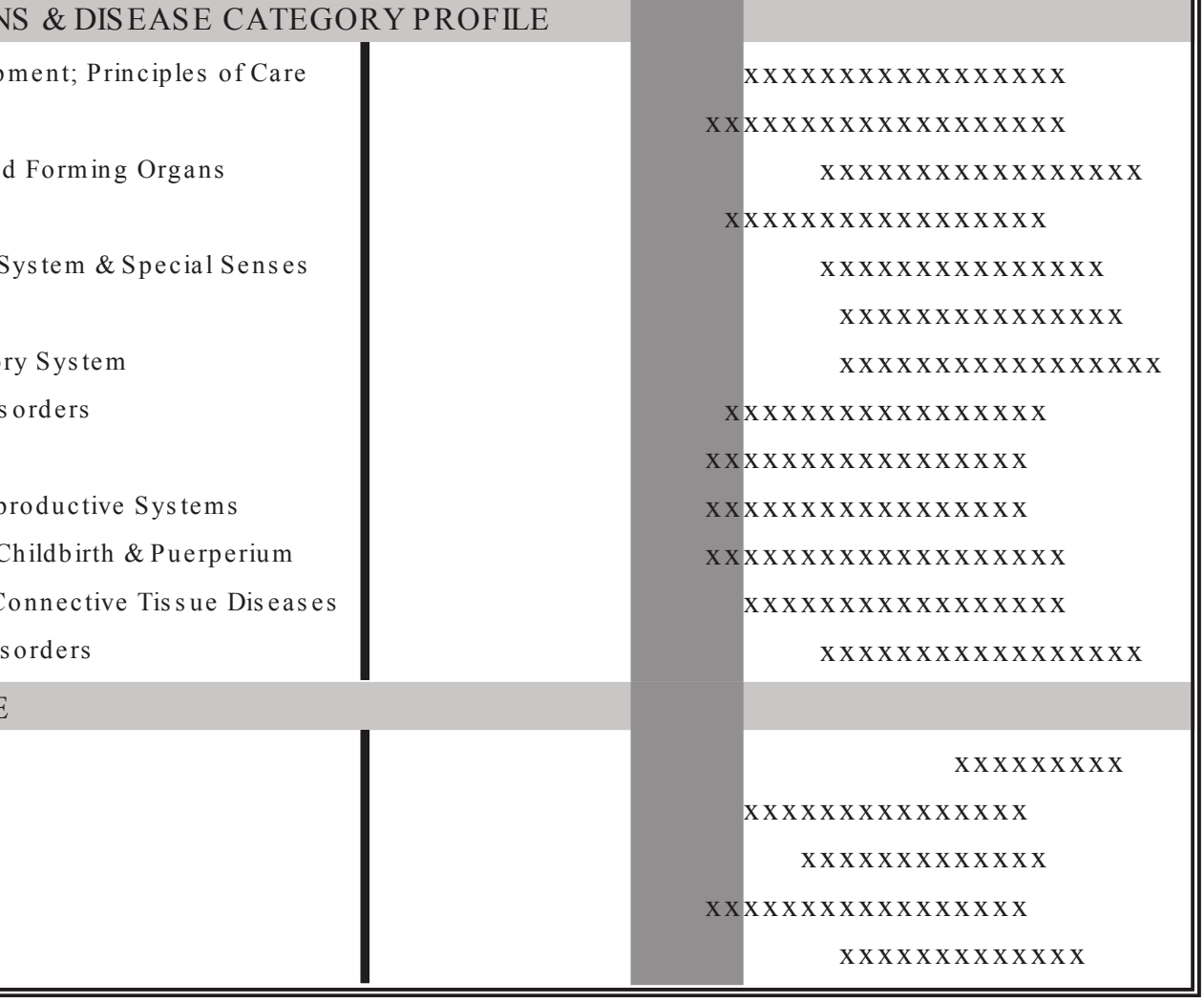
| | |
|---|---|
| 270 | This score is determined by your overall performance on S administrations, the mean and standard deviation for first-time ex Canadian medical schools are approximately 238 and 19, respect falling between 140 and 260. A score of 209 is set by USMLE standard error of measurement (SEM)‡ for this scale is six points. |

§Effective April 1, 2013, test results are reported on a three-digit scale only. Test results reported as passing repre higher on a two-digit scoring scale.

‡Your score is influenced both by your general understanding of clinical science and the specific set of items sele examination. The Standard Error of Measurement (SEM) provides an index of the variation in scores that would examinee were tested repeatedly using different sets of items covering similar content.

**FIGURE 1-1A. Sample score report—front page.**

- **Acing the exam.** Perhaps you are one of those individ ing less than the best will do—and for whom excell examinations is a source of pride and satisfaction. A hig




is provided to aid in self-assessment. The shaded area defines a borderline level of performance for each content comparable to a HIGH FAIL/LOW PASS on the total test.

as of relative strength and weakness. Some bands are wider than others. The width of a performance band reflects narrower bands indicate greater precision. The band width for a given content area is the same for all examinees. performance band extends beyond the displayed portion of the scale. Small differences in the location of bands If two bands overlap, performance in the associated areas should be interpreted as similar. Because Step 2 CK is ny items contribute to more than one content area. As a consequence, caution should be used when interpreting ss content areas.

npared to those from other Step 2 CK administrations.

ing the topics covered in each content area can be found in the USMLE Step 2 CK Content Description and Sample

**ple score report—back page.**

including examination scores related to various special-
t the National Resident Matching Program Web site at
rg under "Match Data."

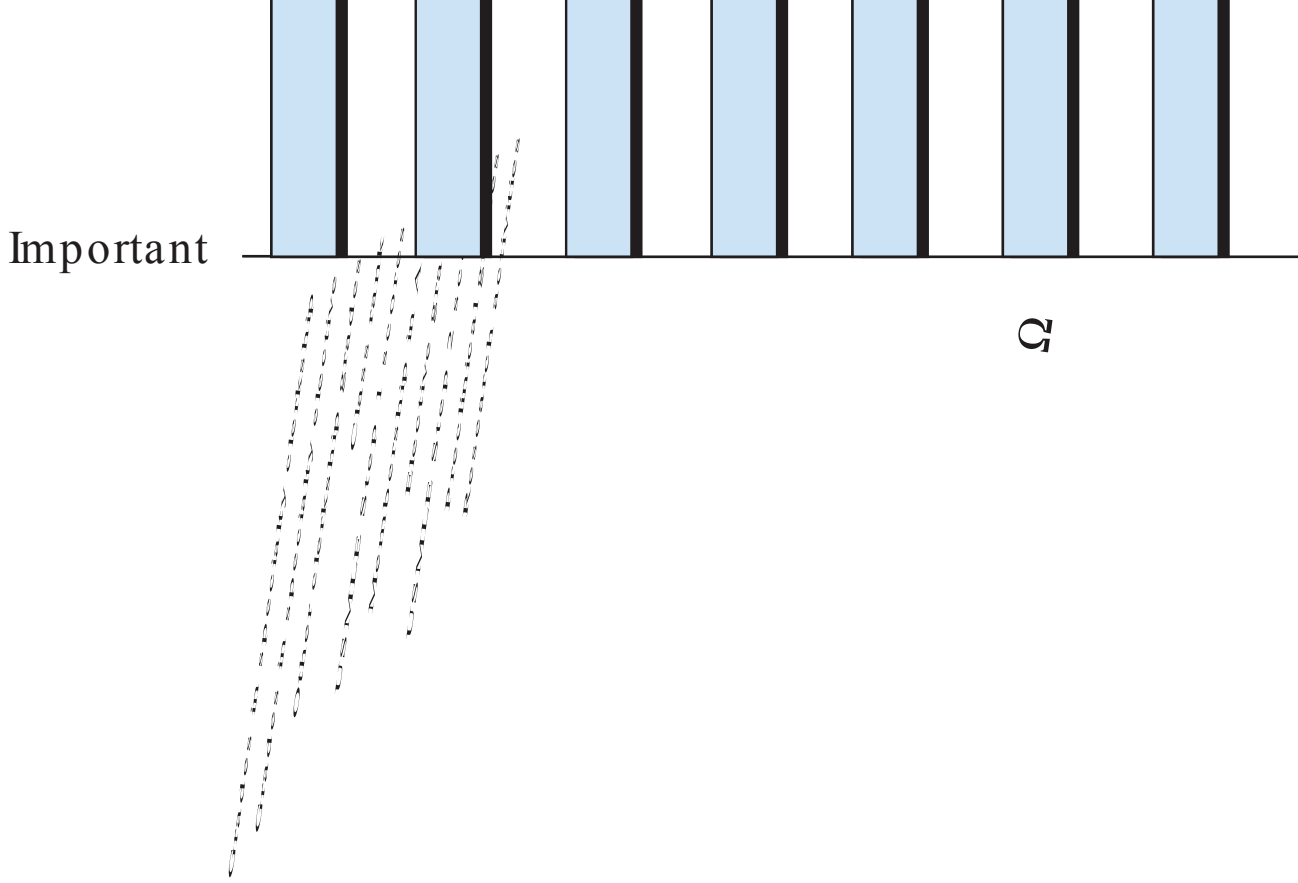



**FIGURE 1-2. Academic factors important to residency directors.**

demonstrate the increasing importance placed on this
dency directors. Some programs are now requiring t
in order to rank candidates for a residency position. I
best interest of candidates to have this examination do
to be available for the residency application. Taking
June or July ensures that scores will be available for th
begins in September. Some programs, however, will
the application process starts. Check with programs i
cialty to determine when to take the examination.

- **Prerequisite to graduation.** If passing the USMLE S
requisite to graduation at your medical school, you w
examination in the fall or winter at the latest.
- **Proximity to clerkships.** Many students feel that the c
rial is fresher in their minds early in the fourth year, r
ment for taking the Step 2 CK earlier in the fall.
- **The nature of your schedule**

nesthesiology book).
uestion books have not been updated to reflect current
p 2 CK.
estion books use poorly written questions, contain factual
xplanations, give overly detailed explanations, or offer no
ll.
rds review is of highly variable quality, may be difficult to
be fraught with bugs.

## BOOKS

are available, so you must decide which ones to buy by
their relative merits. Toward this goal, you should com-
ons from other medical students; read the reviews and rat-
this guide; and examine the various books closely in the
worry about finding the "perfect" book, as many subjects
one.

f review books: those that are stand-alone titles and those
ries. Books in a series generally have the same style, and
hat style is helpful for you and optimal for a given subject.

The bes
the way
review b
using it
may be.

## 

e multiple functions for examinees, including the follow-

tion about strengths and weaknesses in your fund of

ur study schedule.
n form of study.
ng skills.

sible. If you already have your own mnemonics, do not be
orize someone else's. Textbooks are useful, however, to
plete or unclear material.

## COMMERCIAL COURSES

Commercial preparation courses can be helpful for som
offer an effective way to organize study material. Howeve
are costly and require significant time commitment, leav
independent study. Also note that some commercial cour
first-time test takers, students who are repeating the exam
tional medical graduates.

## NBME/USMLE PUBLICATIONS

We strongly encourage students to use the free materials
ing agencies and to study the following NBME publicatio

- USMLE Step 2 Clinical Knowledge (CK): Conte
General Information. This publication provides you
details about the examination (included on the W
.usmle.org; free to all examinees).
- USMLE Step 2 Clinical Knowledge (CK): Sample T
is a PDF version of the test questions and test content
www.usmle.org.
- **NBME Test Delivery Software (FRED) and Tutoria**
valuable practice questions. The questions are avail
from the USMLE Web site. Make sure you are using
FRED and not the older Prometric version.
- **USMLE Web site (http://www.usmle.org).** In additio
become familiar with the CBT format, the sample ite
Web site provide the only questions that are available
makers. Student feedback varies as to the similarity

104-3102

g/contact/
pme.org

104-3190

rg
pme.org

ission for Foreign Medical Graduates (ECFMG)

104-2685

rg/contact.html
.org

Medical Boards (FSMB)
oad, Suite 300

/contact-us
.org

Neurology

Obstetrics

Gynecology

Pediatrics

Psychiatry

Pulmonary

Renal/Genitourinary

Selected Topics in Emergency Me

Rapid Review

**Treatment.** Lists, mnemonics, pull quotes, vignette flash
used when they can help the reader form key association
and black-and-white images are interspersed throughout
of Section 2, we also feature a Rapid Review chapter co
and classic associations that can be studied a day or two be

The content contained herein is useful primarily
reviewing material already learned. The information prese
learning complex or highly conceptual material for the fir

The Database of High-Yield Facts is not comprehensiv
ment your core study material, not as your primary stud
and notes have been condensed and edited to emphasiz
Work with the material, add your own notes and mnemo
that not all memory techniques work for all students.

We update Section 2 biannually to keep current with r
content as well as to expand our database of high-yield inf
we must note that inevitably many other high-yield entrie
yet included in our database.

We actively encourage medical students and faculty to
mnemonics so that we may enhance the database for f
also solicit recommendations of additional study tools th
preparing for the examination, such as diagrams, charts, a
tutorials (see How to Contribute, p. xiii).

## DISCLAIMER

The entries in this section reflect student opinions of
Owing to the diverse sources of material, no attempt has
or reference the origins of entries individually. We have
ics as essentially in the public domain. All errors and omi
corrected if brought to the attention of the authors, eithe
lisher or directly by e-mail.

c o Nd Uc TIo N ABNo RMALITIES

re

N

Y

o PATHY

ATHY

e

omes

ST-ELEVATIo N MYo c ARd IAL INFARc TIo N

AL INFARc TIo N

18 Dyslipidemia

20 Hypertension

21 1° (ESSENTIAL) HYPERTENSIo N

21 2° HYPERTENSIo N

21 HYPERTENSIVE c RISES

22 Pericardial Disease

24 PERIc ARd ITIS

27 c ARd IAc TAMPo NAd E

28 Valvular Heart Disease

28 Vascular Disease

30 Ao RTIc ANEURYSM

30 Ao RTIc d ISSEc TIo N

31 d EEP VENo US THRo MBo SIS

31 PERIPHERAL ARTERIAL d ISEASE

LYMPHEd EMA

32 Syncope

32

33

## Rhythm

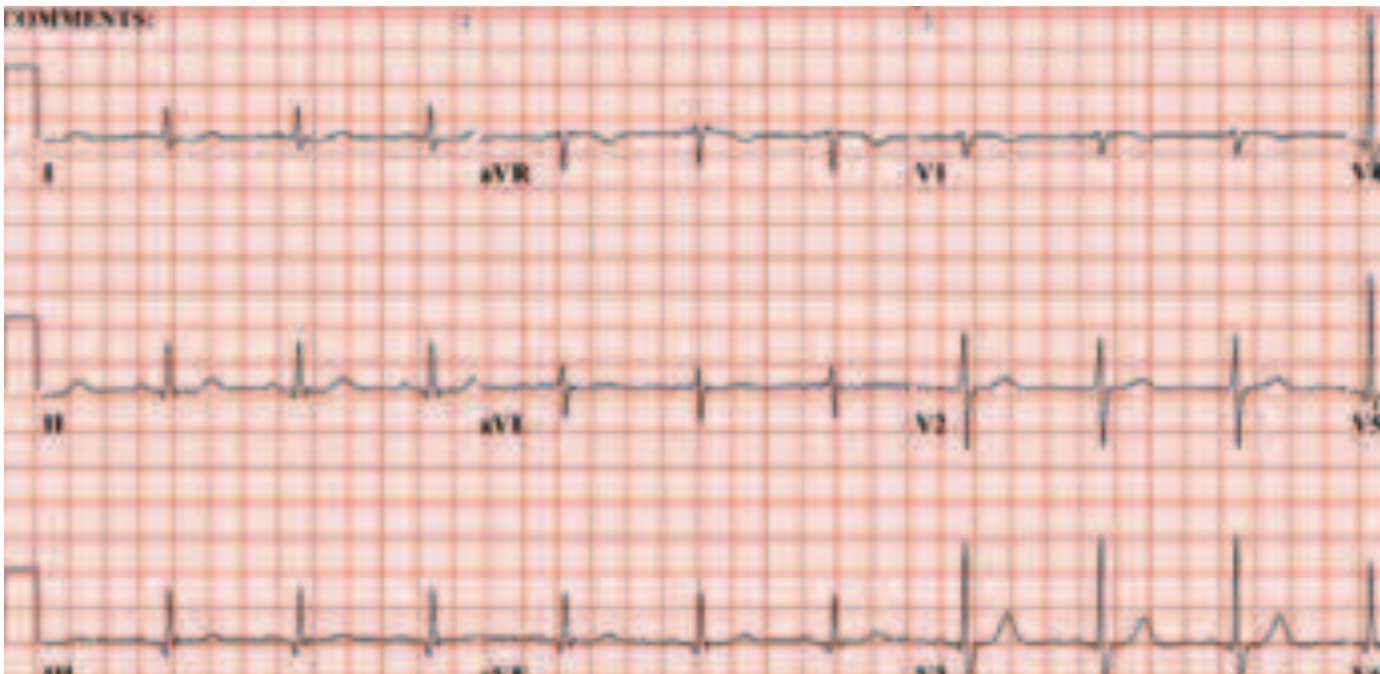
**Sinus rhythm:** Normal rhythm that originates from si
before every QRS and QRS after every P).

## Axis

Can be determined by examining the QRS in leads I and
can be a sign of ventricular hypertrophy or bundle branc
2.1-1.

## Intervals

- **PR interval:** Normally between 120 and 200 msec; in
  val > 200 msec (AV block can also present as P with no
- **QRS interval:** Normally < 120 msec. Two important
  widened QRS are left and right bundle branch block:
  - **Left bundle branch block (LBBB):** QRS duration
    wave and no R wave in $V_1$; wide, tall R waves in I,
    ure 2.1-2).
  - **Right bundle branch block (RBBB):** QRS duratio
    complex ("rabbit ears"); qR or R morphology with a
    QRS pattern with a wide S wave in I, $V_5$, and $V_6$ (se



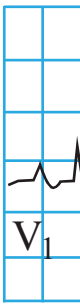
ormally QTc (the QT interval corrected for extremes in
40 msec. In long QT syndrome, QTc > 440 msec. Long
an underdiagnosed congenital disorder that is one cause
d predisposes to ventricular tachyarrhythmias.

Natural progression starts with T-wave inversion, progresses
anges (either depression or elevation), and finally results in
sec or more than one-third of the QRS amplitude) on the
f this, Q waves signify either acute or prior ischemic events
de information on when an event took place.
gression in precordial waves can also be a sign of ische-
is not specific. In a normal ECG, R waves increase in size
S wave between leads $V_1$ and $V_5$. Poor R-wave progres-
inished R waves in these precordial leads.

ent:
**abnormality (P pulmonale):** The P-wave amplitude in
5 mm.
**bnormality (P mitrale):** The P-wave width in lead II is
or terminal ⊖ deflection in $V_1$ is > 1 mm in amplitude
c in duration. Notched P waves can frequently be seen in

**hypertrophy (LVH;** see Figure 2.1-4):
**de of S in $V_1$ + R in $V_5$ or $V_6$ is > 35 mm.**
**riteria:** The amplitude of R in aVL + S in $V_3$ is > 28 mm
20 mm in women.
**r hypertrophy (RVH):** Right-axis deviation and an R wave

**FIGUR**
**block.** C
seen in l
with permi




A




B

**FIGUR**
**branch**
ings are
(Adapted w

P **Pulmo**
P **Mitral**

Often seen in **cardiac tamponade** and **constrictive pe**

- **Systolic murmurs** (see Table 2.1-2 and Figures 2.1-5 a
  - **Aortic stenosis:** A **harsh systolic ejection** murmur carotids.
  - **Mitral regurgitation:** A **holosystolic** murmur that r
  - **Mitral valve prolapse:** A midsystolic or late systo **preceding click.**
  - **Flow murmur:** Usually a soft murmur that is posit common, and does not imply cardiac disease).
- **Diastolic murmurs** (see Table 2.1-2 and Figures 2.1- abnormal.
  - **Aortic regurgitation:** An **early decrescendo** murmu
  - **Mitral stenosis:** A **mid- to late, low-pitched** murm
- **Gallops:**
  - **S3 gallop:** A sign of fluid overload (ie, heart failu ease); often normal in younger patients and in hig pregnancy).
  - **S4 gallop:** A sign of decreased compliance (ie, hy dysfunction); usually pathologic but can be normal and in athletes.
- **Edema:**
  - **Pulmonary:** Left heart failure (fluid "backs up" into
  - **Peripheral:** Right heart failure and biventricular up" into the periphery). Also nephrotic syndrom lymphedema, hypoalbuminemia, and drugs.
- **Peripheral pulses:**
  - **Increased:** Compensated aortic regurgitation (bou tation (greater in arms than legs); patent ductus arte
  - **Decreased:** Peripheral arterial disease; late-stage he
  - **Pulsus paradoxus** (↓ systolic BP with inspiration **ponade;** also seen in obstructive lung diseases, ten and foreign body in airway.
  - **Pulsus alternans** (alternating weak and strong pu

1-5 outline the etiologies, clinical presentation, and treat-
praventricular and ventricular tachyarrhythmias.

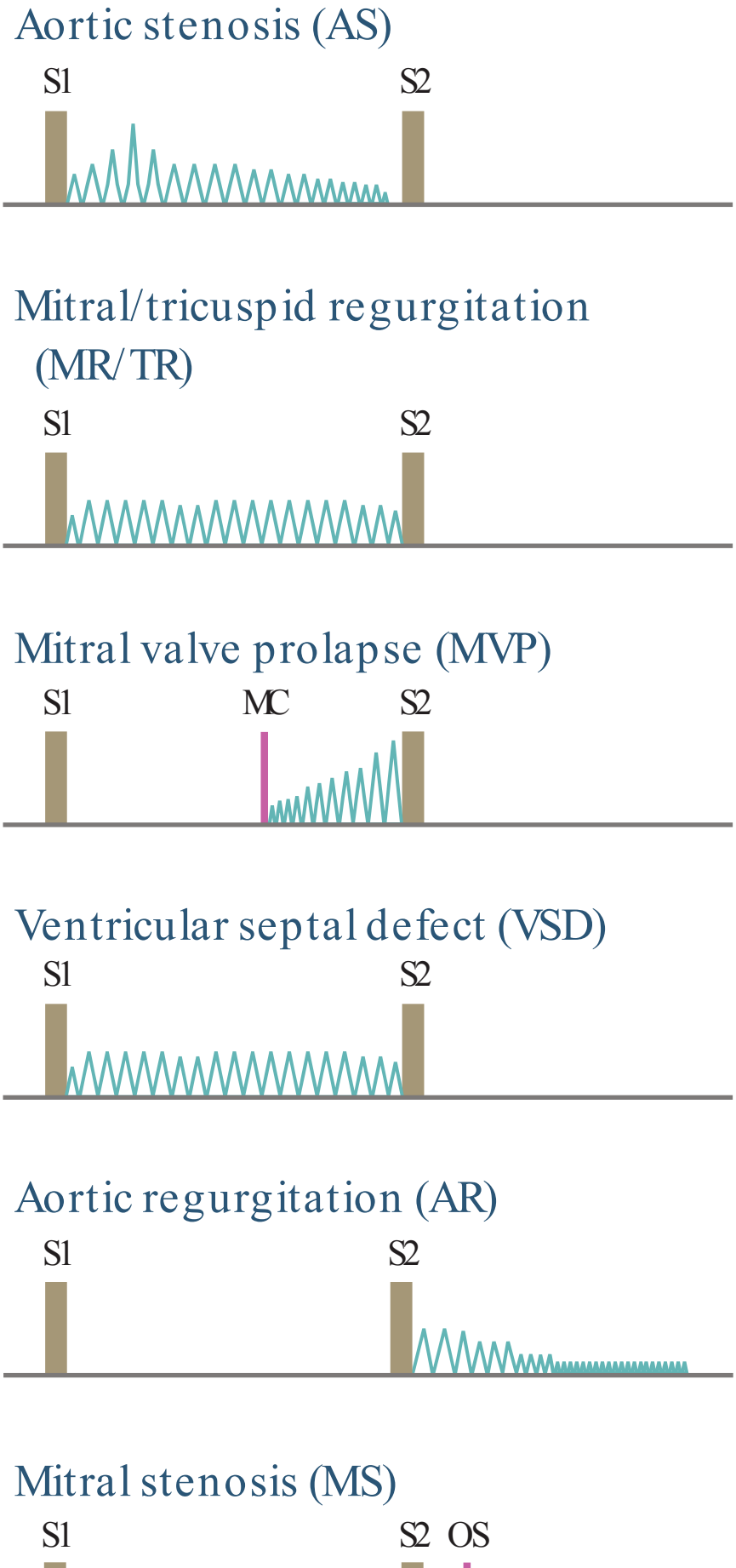



**FIGURE**
**tions.** A
associate
mitral va
cuspid v
Le Tet al. P
NY: McGra

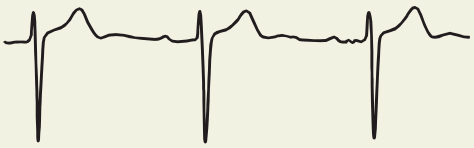
Manag
fibrill t

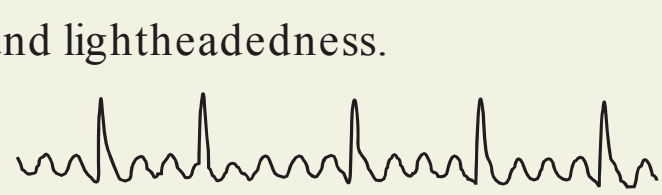
**ABCD**

**A**nticoag
**β**-blocke
**C**ardiove
**D**igoxin

Use the
system t
fibrillatio
of 2 or m

- **CHF**
- **HTN**
- **Age**
- **Diab**
- **Stro**
- **Vas**

| | | |
|---|---|---|
| occur in normal iduals; associated ↑ vagal tone and β-blocker or CCB | Asymptomatic.  | PR interval > 200 msec. |
| ef ects (digoxin, ckers, CCBs) or **gal tone;** right nary ischemia or ction. | Usually asymptomatic.  | Progressive PR lengthening until a dropped beat occurs; the PR interval then resets. |
| lts from fib otic se of the uction system or acute, subacute, ior MI. | Occasionally syncope; **frequent progression to third-degree AV block.**  | Unexpected dropped beat(s) without a change in PR interval. |
| ectrical munication een the atria and ricles. | Syncope, dizziness, acute heart failure, hypotension, cannon A waves.  | No relationship between P waves and QRS complexes. |
| terogeneous der that leads ermittent aventricular varrhythmias and | 2° to tachycardia or bradycardia; may include syncope, palpitations, dyspnea, chest pain, TIA, and stroke. | |

| | | |
|---|---|---|
| volume contraction, nfection, or PE. | | |
| **Acute AF—PIRATES:**<br>**P**ulmonary disease<br>**I**schemia<br>**R**heumatic heart disease<br>**A**nemia/**A**trial myxoma<br>**T**hyrotoxicosis<br>**E**thanol<br>**S**epsis<br>**Chronic AF**—HTN, CHF. | Often asymptomatic but may present with shortness of breath, chest pain, or palpitations. Physical examination reveals an **irregularly irregular pulse.**<br>  | No discernible P wave with variable and irregular QRS respons |
| Circular movement of electrical activity around the atrium at a rate of approximately 300 times per minute. | Usually asymptomatic but can present with palpitations, syncope, and lightheadedness.<br>  | Regular rhythm; **"sawtooth" appeara of P waves** can be see The atrial rate is usual 240–320 bpm and the ventricular rate ~ 150 bpm. |
| Multiple atrial pacemakers or reentrant pathways; | May be asymptomatic. At least **3 dif erent P-wave morphologies.** | Three or more unique P-wave morphologies rate > 100 bpm. |

| | | | |
|---|---|---|---|
| nearly<br>ously. | | | a<br>a |
| c<br>n between<br>n and<br>hat causes<br>circuit.<br>PW. | Palpitations, shortness of breath, angina, syncope, lightheadedness. | A retrograde P wave is often seen after a normal QRS. A **preexcitation delta wave is characteristically seen in WPW.** | S |
| opic<br>er in the<br>ot sinus | Palpitations, shortness of breath, angina, syncope, lightheadedness. | Rate > 100 bpm; P wave with an unusual axis **before** each normal QRS. | A<br>t<br>a<br>d |

including that of the New York Heart Association (NY
tional severity, left-sided vs right-sided failure, and systoli
ure (see Tables 2.1-6–2.1-8).

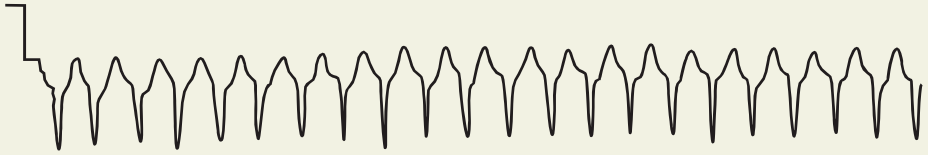
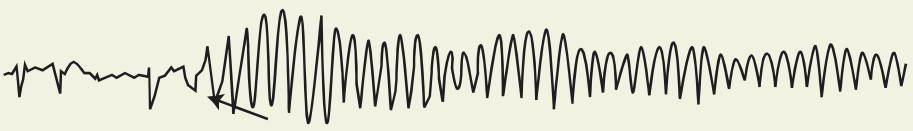
nt-sided
ilure.

## SYSTOLIC DYSFUNCTION

A ↓ **EF (< 50%)** and ↑ left ventricular end-diastolic volu
inadequate left ventricular contractility or ↑ afterload. The
for ↓ EF and ↑ preload through hypertrophy and ventricu
Starling law), but the compensation ultimately fails, lead
work and worsening systolic function.

### History/Pe

- **Exertional dyspnea is the earliest and most commo**
  **tom** and progresses to orthopnea, paroxysmal noctur

| | | |
|---|---|---|
| yroidism. | | |
| al fast<br>ry pathway<br>ia to<br>e. | Palpitations, dyspnea, dizziness, and rarely cardiac death. | Characteristic delta wave with widened Q complex and shorten PR interval (see Figur 2.1-7). |
| associated<br>D, MI, and<br>al heart | Nonsustained VT (lasts < 30 seconds) is often asymptomatic; sustained VT (lasts > 30 seconds) can lead to palpitations, hypotension, angina, and syncope. Can progress to VF and death.<br>  | Three or more consecutive PVCs; wi QRS complexes in a regular rapid rhythm may see AV dissociat |
| ed with<br>d structural<br>sease.<br>ociated<br>diac arrest<br>er with<br>). | Syncope, absence of BP, no pulse.<br>  | Totally erratic wide-complex tracing. |
| ed with<br>syndrome,<br>ythmic<br>e to<br>ions,<br>emia, | Can present with sudden cardiac death; typically associated with palpitations, dizziness, and syncope.<br>  | Polymorphous QRS; V with rates between 1 and 250 bpm. |

achyar-
vave
short-
uced with
SMLEStep 1

ke it in.

## Treatment

- **Acute:**
  - **Pharmacologic therapy** (see Table 2.1-9):
    - Loop diuretics (most commonly) for **aggressive**
    - ACEIs for patients who can tolerate (consider a blockers [ARBs] for those who can't).
    - β-blockers should be avoided during decom should be restarted once patient is euvolemic.
  - Correct underlying causes such as arrhythmias, m and drugs (eg, CCBs, antiarrhythmics, NSAIDs, a valvular disease, high-output states).
- Treat acute pulmonary congestion with **LMNOP** (see
- **Chronic:**
  - **Lifestyle:** Control comorbid conditions, and **limit fluid intake.**
  - **Pharmacologic therapy:**
    - **β-blockers and ACEIs/ARBs:** Help prevent rem and ↓ mortality for NYHA class II–IV patients worsen edema).
    - **Diuretics** (most commonly loop diuretics): Pr load.
    - Low-dose **spironolactone:** Shown to ↓ morta with NYHA class III–IV heart failure.
    - **Daily ASA and a statin** are recommended if th is a prior MI.
- **Advanced treatments:**
  - **Implantable cardiac defibrillator (ICD)** in patients
  - **Left ventricular assist device (LVAD) or cardiac tra** necessary in patients who are unresponsive to maxima

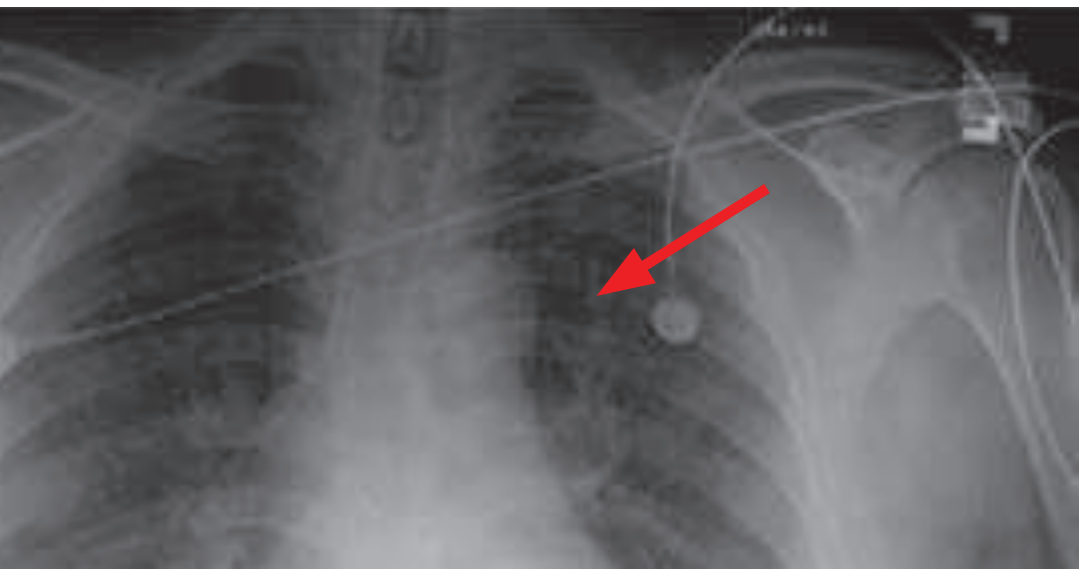
| | | |
|---|---|---|
| | Pulmonary congestion, cardiomegaly. | Pulmonary congestion |
| y | Q waves, ↓ EF (< 40%). | LVH, normal EF (> 55% |

## FUNCTION

**ricular compliance with normal systolic function.** The impaired active relaxation (2° to ischemia, aging, and/or paired passive filling (scarring from prior MI; restrictive eft ventricular end-diastolic pressure ↑, cardiac output ormal, **and EF is normal or ↑.**

ble and unstable angina, shortness of breath, dyspnea on as, MI, heart failure, and sudden death.

t-line therapy (see Table 2.1-9).
d BP control via β-blockers, ACEIs, ARBs, or CCBs.
seful in these patients.



Aman
exacer
is now

| | |
|---|---|
| **ronolactone,** triamterene, amiloride | Hyperkalemia, gynecomastia, se |
| tazolamide | Hyperchloremic metabolic acido toxicity, sulfa allergy. |
| nitol | Pulmonary edema, dehydration anuria and CHF. |

# Cardiomyopathy

Myocardial disease; categorized as dilated, hypertrophic Table 2.1-10 and Figure 2.1-9).

## DILATED CARDIOMYOPATHY

The most common cardiomyopathy. **Left ventricular** must be present for diagnosis. Most cases are idiopathic, b include alcohol, myocarditis, postpartum status, drugs ( cocaine), endocrinopathies (thyroid dysfunction, acromeg toma), infection (**coxsackievirus,** HIV, **Chagas disease,** pa

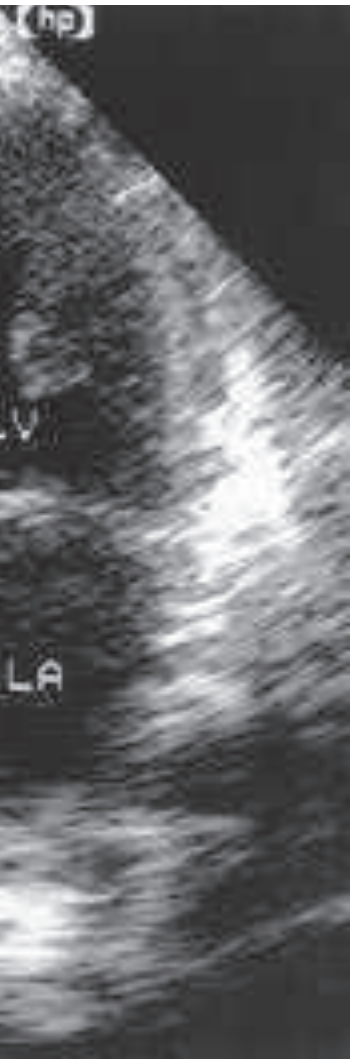
**TABLE 2.1-10. Differential Diagnosis of Cardiomyopathies**

| | Type | |
|---|---|---|
| **Variable** | **dilated** | **hypertrophic** |
| Major abnormality | Impaired contractility | Impaired relaxation |





B




**diomyopathies.** Echocardiogram four-chamber views of **(A)** a normal myopathy, and **(C)** hypertrophic cardiomyopathy. (Reproduced with permis-
ne Heart, 12th ed. New York, NY: McGraw-Hill; 2008.)

disorders (wet beriberi). The most common causes of 2° athy are ischemia and long-standing hypertension.

ith gradual development of CHF symptoms.
en reveals displacement of the left ventricular impulse,

An S3 ga
filling in
and is as
cardiom

### History/Pe

- Patients are often asymptomatic but may also present
  nea, palpitations, angina, or sudden cardiac death.
- Key finding is **a systolic ejection crescendo-decresc**
  ↑ **with** ↓ **preload** (eg, Valsalva maneuver, standing) a
  (eg, passive leg raise). Examination also often reveal
  impulse, and an S4 gallop.

### Diagnosis

- **Echocardiography is diagnostic** and shows an asymm
  septum and dynamic obstruction of blood flow.
- ECG may show signs of LVH.
- CXR may reveal left atrial enlargement (LAE) 2° to mi

### Treatment

- β-blockers are initial therapy for symptomatic relief; C
  agents.
- Surgical options for HOCM with persistent symptoms
  sion or alcohol ablation of the myocardial septum.
- Patients should avoid intense athletic competition and

## RESTRICTIVE CARDIOMYOPATHY

Decreased elasticity of myocardium leading to impaired d
out significant systolic dysfunction (a normal or near-norm
**by infiltrative disease** (amyloidosis, sarcoidosis, hemochr
ring and fibrosis (2° to radiation).

### History/Pe

Signs and symptoms of left-sided and right-sided heart
symptoms of right-sided heart failure (JVD, peripheral ede

**ude:**
litus (DM)
y of premature CAD (males < 55, females < 65)

besity

45, females > 55)

alents include DM, symptomatic carotid artery disease, isease, and abdominal aortic aneurysm (AAA).

**ain 2° to myocardial ischemia** ($O_2$ supply-and-demand etal (variant) angina mimics angina pectoris but is caused onary vessels. It classically affects young women at rest in nd is associated with ST-segment elevation in the absence levation.

l consists of **substernal chest pain** that is usually **precipi-** exertion and is **relieved by rest** or nitrates.
to neck or arm and may be associated with shortness of omiting, diaphoresis, or lightheadedness.
escribed as dull and pressure-like.

initial test for any type of chest pain.
T-segment or wall-motion changes with exercise or phar- are diagnostic of CAD. ECG stress test is contraindicated

Major ris
male ge
family h

Pain tha
changes
touch is

that leads to acute myocardial ischemia.

## UNSTABLE ANGINA/NON-ST-ELEVATION MYOCARDIAL INF

Unstable angina is defined as chest pain that is (1) **new or ing** (ie, occurs with less exertion, lasts longer, or is less re tions), or (3) **occurs at rest;** it is distinguished from stable patient history. It signals the presence of possible **impendi** on plaque instability. In contrast, **NSTEMI** indicates sis marked by elevations in **troponin I and creatine kin (CK-MB)** without ST-segment elevations seen on ECG.

### Diagnosis

- Patients should be risk stratified according to the Thro dial Infarction (TIMI) study criteria (see Table 2.1-11)
- Unstable angina is not associated with elevated cardi changes may be seen on ECG.
- NSTEMI is diagnosed by serial cardiac enzymes and E

### Treatment

- Treat acute symptoms with ASA, $O_2$, IV nitroglycerin and consider β-blockers as hemodynamics allow.

**TABLE 2.1-11. TIMI Risk Score for Unstable Angina/NSTEMI**

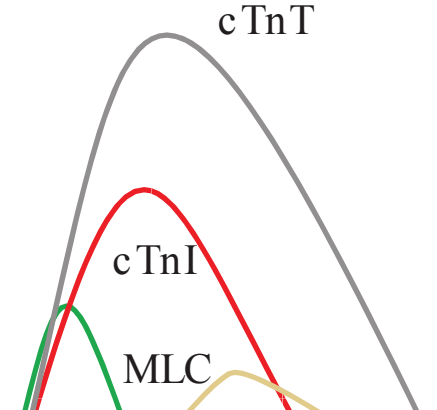
| Characteristics |
|---|
| **History** |
| Age ≥ 65 years |
| Three or more CAD risk factors (premature family history, DM, smok ↑ cholesterol) |

sis.

**cute-onset substernal chest pain,** commonly described as
tness that can radiate to the left arm, neck, or jaw.
**ptoms:** Diaphoresis, shortness of breath, lightheadedness,
vomiting, and syncope.
**ation:** May reveal arrhythmias, hypotension (cardiogenic
ence of new CHF.
**tor of survival is left ventricular EF.**

ST-segment elevations or new LBBB. ST-segment depres-
ant R waves in leads $V_1$–$V_2$ can also be reciprocal change
ior wall infarct.
**CG changes:** Peaked T waves → ST-segment elevation
-wave inversion → ST-segment normalization → T-wave
ver several hours to days.
**s:**
s the most sensitive and specific cardiac enzyme.
the CK-MB/total CK ratio (CK index) are also regularly

n I and CK-MB can take up to 6 hours to rise following
hest pain (see Figure 2.1-10).




When y
from a

**MOAN**

**M**orphin
**O**xygen
**A**SA
**N**itrogen

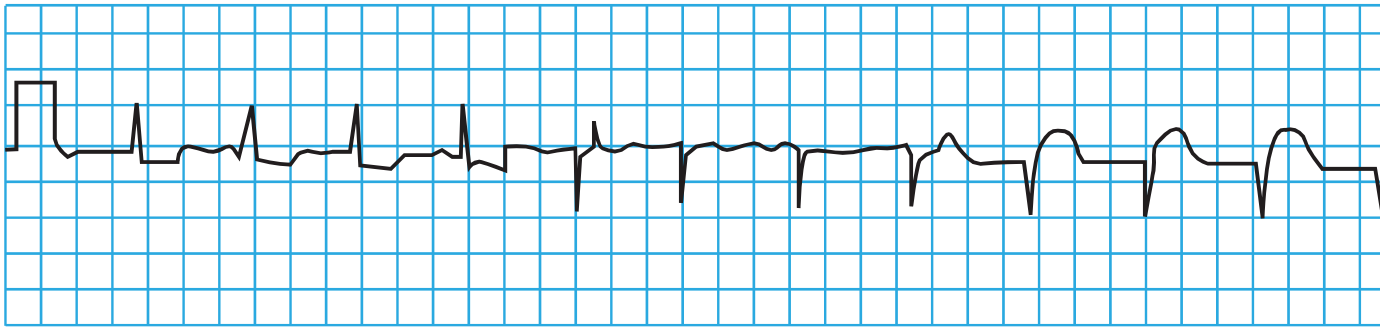
Women
post–he
have aty

## Treatment

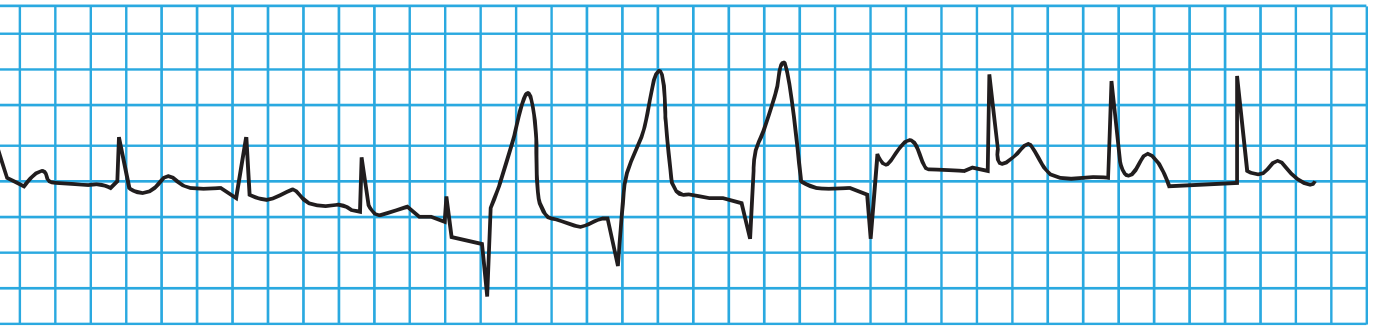
- **Initial treatment:**
  - **Key medications:** ASA, β-blockers, clopidogrel, and $O_2$.
  - If the patient is in heart failure or in cardiogenic β-blockers; instead, give ACEIs provided that the tensive.
  - In inferior wall MI, avoid nitrates due to risk of seve
- **Interventions:**
  - **Emergent angiography and PCI** should be perform
  - If PCI cannot be performed within 90 minutes, **an** traindications to thrombolysis (eg, a history of he recent ischemic stroke, severe heart failure, or card the patient presents within 3 hours of chest pain ons tPA, reteplase, or streptokinase should be performed
- Long-term treatment includes ASA, ACEIs, β-blocke (goal LDL < 100 mg/dL), and clopidogrel (if PCI was risk factors with dietary changes, exercise, and tobacco

## Complications

- **Arrhythmia** is the most common complication and m of death following acute MI.







**terior wall myocardial infarction.** This patient presented with acute owed acute ST-segment elevation in leads aVL and $V_1$–$V_6$, and hyper-

complications include reinfarction, left ventricular wall ericarditis, papillary muscle rupture (with mitral regurgita- ular aneurysm or pseudoaneurysm, and mural thrombi.
mmon post-MI complications:
art failure.
hythmia, pericarditis.
eft ventricular wall rupture (acute pericardial tamponade rical alternans, pulseless electrical activity), papillary mus- evere mitral regurgitation).
**nths:** Ventricular aneurysm (CHF, arrhythmia, persistent elevation, mitral regurgitation, thrombus formation).
**me,** an autoimmune process occurring 2–10 weeks post-MI, er, pericarditis, pleural effusion, leukocytosis, and ↑ ESR.

## mia

vel > 200 mg/dL, LDL > 130 mg/dL, triglycerides > 150 40 mg/dL, all of which are risk factors for CAD. Etiolo- , DM, alcoholism, hypothyroidism, nephrotic syndrome, shing syndrome, OCP use, high-dose diuretic use, and sterolemia.

**statin.**

- For patients with LDL between 70 and 189 mg/dL
  - ≥ 7.5% 10-year risk → **high-intensity statin.**
  - Between 5% and 7.5% 10-year → **moderate-inte**
  - ≤ 5% 10-year risk → **no statin.**
- For patients with LDL between 70 and 189 mg/dL
  - ≥ 7.5% 10-year risk → **high-intensity statin.**
  - ≤ 7.5% 10-year → **moderate-intensity statin.**
- For patients with LDL ≥ 190 mg/dL → **high-intens**
- High-intensity therapy is defined as a goal reductio moderate-intensity as lowering by 30–50% or by and dosing guidelines (ie, atorvastatin 80 mg).

- As you cannot calculate the patient's risk on the USM ous signs of elevated risk (smoking, diabetes) or dec healthy) when deciding if statin therapy is appropriate.
- The **first intervention** should be a **12-week trial of di** patient with no known atherosclerotic vascular diseas lipid-lowering agents are listed in Table 2.1-12.

**ng Agents**

| Examples | Effect on lipid profile | Si |
|---|---|---|
| Atorvastatin, simvastatin, lovastatin, pravastatin, rosuvastatin | ↓ LDL, ↓ triglycerides | ↑ LFTs, myositis, |
| Gemfibozil | ↓ triglycerides, ↑ HDL | GI upset, choleli |
| Ezetimibe | ↓ LDL | Diarrhea, abdon angioedema. |

no identifiable cause. Represents 95% of cases of HTN. a family history of HTN or heart disease, a high-sodium ty, ethnicity (blacks > whites), and advanced age.

matic until complications develop.
be evaluated for end-organ damage to the brain (stroke, cotton-wool exudates, hemorrhage), heart (LVH), and kid-, CKD). Renal bruits may signify renal artery stenosis as N.

BUN/creatinine, and electrolytes to assess the extent of nd possible 2° causes.

**yle** modifications (weight loss, exercise, and diet improve-decreased alcohol and salt intake).
ry by age and comorbidities.
, ACEIs, and β-blockers have been shown to ↓ mortality d HTN. They are first-line agents unless a comorbid con-nother medication (see Table 2.1-14).
for end-organ complications, including renal complica-tinine, urine protein-to-creatinine ratio) and cardiac com-evidence of hypertrophy).

**8 Classifi ation and Management of Hypertension**

| ulation | initiate treatment at: | treat to goal of: |
|---|---|---|
| | SBP ≥ 150 mm Hg | SBP ≤ 150 mm Hg |

Treatm

**ABCD**

ACEIs/AI
β-blocke
CCBs
Diuretics

Awon

| | |
|---|---|
| Chronic kidney disease | **ACEIs, ARBs** |
| BPH | Diuretics, $\alpha_1$-adrenergic blockers |
| Isolated systolic HTN | Diuretics, ACEIs, CCBs (dihydropyridines) |
| Pregnancy | β-blockers (typically labetalol), hydralazin |

## 2° HYPERTENSION

Hypertension 2° to an identifiable organic cause. See T
diagnosis and treatment of common causes.

## HYPERTENSIVE CRISES

A spectrum of clinical presentations in which elevated BP
mm Hg) lead to end-organ damage.

### History/Pe

Present with end-organ damage revealed by acute kidne
(ischemia or MI), back pain (aortic dissection), or chan
(hypertensive encephalopathy).

### Diagnosis

- **Hypertensive urgency:** Elevated BP with **mild to n**
**(headache, chest pain) without end-organ damage.**
- **Hypertensive emergency:** Elevated BP with **signs**
**impending end-organ damage** such as acute kidney
hemorrhage, papilledema, or ECG changes suggestive

| | | |
|---|---|---|
| | | of the ef erent arteriole option if angioplasty is |
| | Common in women > 35 years of age, obese women, and those with long-standing use. | Discontinue OCPs (ef ect |
| | An adrenal gland tumor that secretes epinephrine and norepinephrine, leading to **episodic headache, sweating, and tachycardia.** | Diagnose with urinary m catecholamine levels or p removal of tumor after **tr and β-blockers.** |
| | Most often 2° to an aldosterone-producing adrenal adenoma. Causes the **triad of HTN, unexplained hypokalemia, and metabolic alkalosis.** | Metabolic workup with p level; ↑ aldosterone and hyperaldosteronism. Sur |
| | Due to an ACTH-producing pituitary tumor, an ectopic ACTH-secreting tumor, or cortisol secretion by an adrenal adenoma or carcinoma. Also due to exogenous steroid exposure. (See the Endocrinology chapter for more details.) | Surgical removal of tumo steroids. |
| ta | See the Pediatrics chapter. | Surgical repair. |

## al Disease

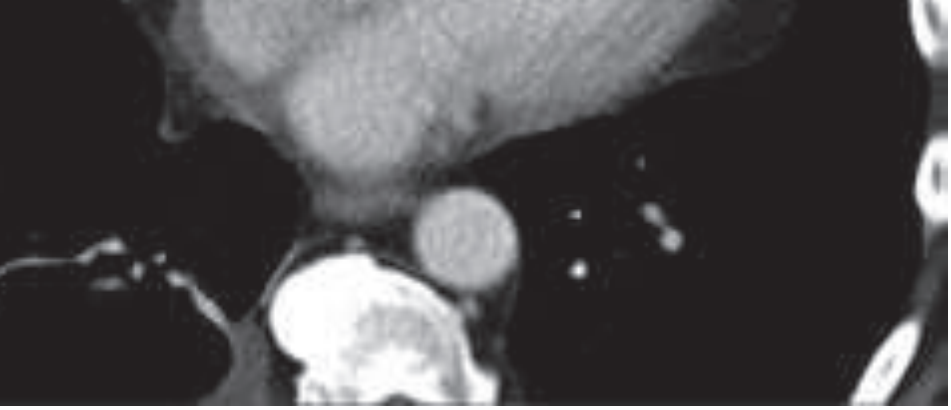
or chronic pericardial insults; may lead to pericardial effu-



Causes

| | | |
|---|---|---|
| | | depression. |
| ril, enalapril, fosinopril, pril, lisinopril | Block aldosterone formation, reducing peripheral resistance and salt/water retention. | Cough, angio leukopenia, h |
| n, valsartan, irbesartan | Block aldosterone ef ects, reducing peripheral resistance and salt/water retention. | Rashes, leuko hyperkalemia |
| pyridines (nifedipine, ne, amlodipine), ydropyridines (diltiazem, nil) | ↓ smooth muscle tone and cause vasodilation; may also ↓ cardiac output. | **Dihydropyri** flushin , pe **Nondihydrop** ↓ contracti |
| zine, minoxidil | ↓ peripheral resistance by dilating arteries/arterioles. | **Hydralazine:** syndrome. **Minoxidil:** O |
| n, terazosin, ybenzamine | Cause vasodilation by blocking actions of norepinephrine on vascular smooth muscle. | Orthostatic h |
| lopa, clonidine | Inhibit the sympathetic nervous system via central $\alpha_2$-adrenergic receptors. | Somnolence, hypotension, HTN. |

- **Examination:** May reveal a pericardial friction rub. El sus paradoxus (a ↓ in systolic BP > 10 mm Hg on insp ent with tamponade.

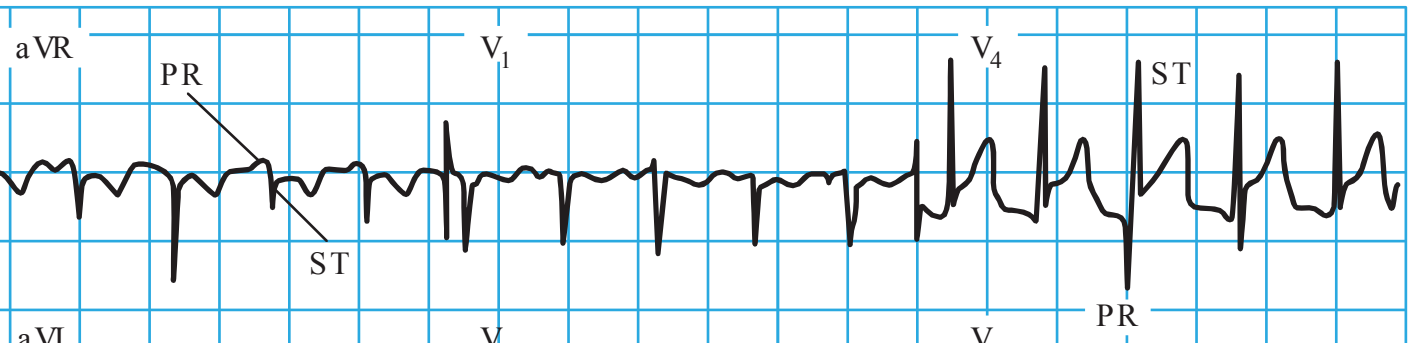


**diographic findings in pericarditis** Contrast enhanced CT at the lar septum demonstrates a small pericardial effusion, with thickening ent of the pericardium consistent with infection in this post-surgical g the pericardium anteriorly is the result of dehiscence of the median th permission from USMLE-Rx.com.)

viral pericarditis). Avoid corticosteroids within a few days can predispose to ventricular wall rupture.
ions without symptoms can be monitored, but evidence equires pericardiocentesis with continuous drainage as

## ADE

**pericardial sac,** leading to compromised ventricular fill-utput. The rate of fluid formation is more important than sion. Risk factors include pericarditis, malignancy, SLE, mmonly stab wounds medial to the left nipple).



large effusion (see Figure 2.1-15).
- If present on ECG, **electrical alternans is diagnostic** o effusion.

### Treatment

- Aggressive volume expansion with IV fluids.
- Urgent pericardiocentesis (aspirate will be nonclotting
- Decompensation may warrant pericardial window.

## Valvular Heart Disease

Until recently, rheumatic fever (which affects the mitra than the aortic valve) was the most common cause of valv US adults; the leading cause is now mechanical degene listed in Table 2.1-17 along with their etiologies, presenta treatment.

## Vascular Disease

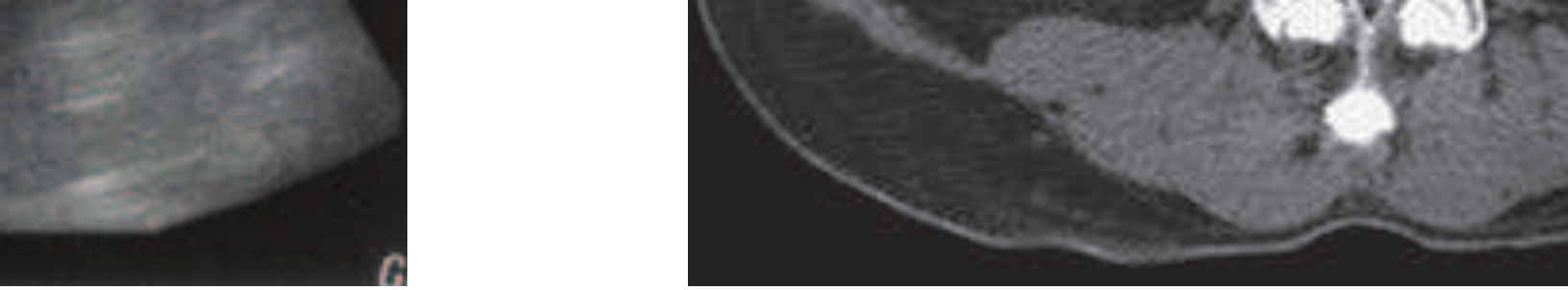
### AORTIC ANEURYSM

Greater than 50% dilatation of all three layers of the aorti rysms are most commonly **associated with atherosclerosi** nal and > 90% originate below the renal arteries.

### History/Pe

- **Usually asymptomatic** and discovered incidentally radiologic study.
- Examination demonstrates a **pulsatile abdominal m**

| | | |
|---|---|---|
| adolescence. | within 5 years.<br>**Sx** (also indications for valve replacements): **ACS**—**A**ngina, **C**HF, **S**yncope. | carotids.<br>**Dx:** Echocardiography. |
| **cute:** Infective endocarditis, aortic dissection, chest trauma.<br>**hronic:** Valve malformations, rheumatic fever, connective tissue disorders. | **Acute:** Rapid onset of pulmonary congestion, cardiogenic shock, and severe dyspnea.<br>**Chronic:** Slowly progressive onset of dyspnea on exertion, orthopnea, and PND. | **PE: Blowing diastolic murmur** at the left sterna border, mid-diastolic rumble (Austin Flint murmur), and midsystolic apical murmur.<br>**Widened pulse pressure** causes de Musset sign (head bob with heartbeat), Corrigan sign (water-hammer pulse), and Duroziez sign (femoral bruit).<br>**Dx:** Echocardiography. |
| most common etiology inues to be **rheumatic r.** | Symptoms range from dyspnea, orthopnea, and PND to infective endocarditis and arrhythmias. | **PE:** Opening snap and **mid-diastolic murmur** at the apex; pulmonary edema.<br>**Dx:** Echocardiography. |
| arily 2° to **rheumatic r or chordae ineae rupture after** nfective endocarditis. | Patients present with dyspnea, orthopnea, and fatigue. | **PE: Holosystolic murmur** radiating to the axilla.<br>**Dx:** Echocardiography will demonstrate regurgitant |



B

**l aortic aneurysm. (A)** Ultrasound image of an AAA (Ao = aorta). **(B)** Transaxial ima
ysm with extensive mural thrombus (arrowhead). (Image A reproduced with permission from Ti
e, 6th ed. New York, NY: McGraw-Hill; 2004. Image B reproduced with permission from Doherty GM. Current Dia

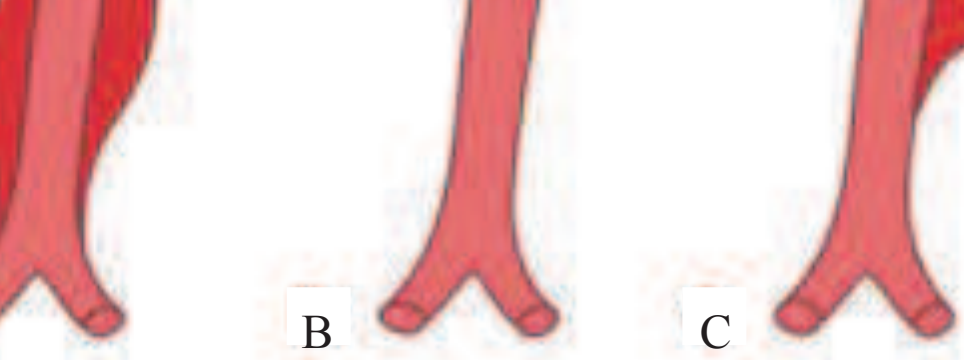
## AORTIC DISSECTION

A transverse tear in the intima of a vessel that results in
media, creating a false lumen and leading to a hematoma
gitudinally. **Most commonly 2° to HTN.** The most com
are above the aortic valve and distal to the left subclavia
occurs at 40–60 years of age, with a greater frequency in m

mmonly

### History/Pe

- **Presentation: Sudden tearing/ripping pain in the ant**
  ing) or back (descending).
- **Examination:**
  - Patients are typically hypertensive. If hypotensive,
    tamponade, hypovolemia from blood loss, or oth
    etiologies.
  - **Asymmetric pulses and BP measurements.**
  - A murmur of aortic regurgitation may be heard i
    involved with a proximal dissection.
  - **Neurologic deficits** may be seen if the **aortic arc**
    are involved.
- Signs of pericarditis or pericardial tamponade may be s




**rtic dissection.** Stanford classification of aortic dissections. Shown cending types and **(C)** descending type. (Reproduced with permission from

involves the ascending aorta, it is a surgical emergency; ctions can often be managed with BP and heart rate con-

## OMBOSIS

e large veins of the extremities or pelvis. The classic **Vir-** factors includes venous stasis (eg, from plane flights, bed nt venous valves in the lower extremities), endothelial , injury to the lower extremities), and hypercoagulable cy, pregnancy, OCP use).

Virchow
(2) traun
(3) hype

ilateral lower extremity pain and swelling.
calf tenderness with passive foot dorsiflexion (poor sensi- city for DVT).

; a spiral CT or V/Q scan may be used to evaluate for PE

A ⊖ D-d

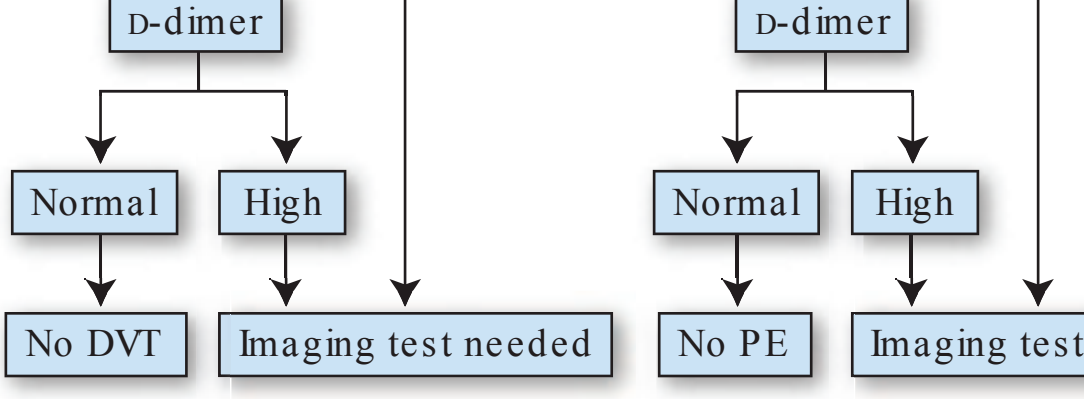



**FIGURE 2.1-18. Algorithm for diagnostic imaging of deep-vein t nary embolism.** (Reproduced with permission from Fauci AS et al. Harrison's Principles of York, NY: McGraw-Hill; 2008.)

## PERIPHERAL ARTERIAL DISEASE

Defined as a restriction of the blood supply to the extrem rotic plaque. The lower extremities are most commonl manifestations depend on the vessels involved, the extent tion, and the presence of collateral blood flow.

### History/Pe

- Presents with intermittent claudication (reproducible with walking and is relieved with rest). As the disea occurs at rest and affects the distal extremities. Dorsal develop 2° to poor perfusion. A painful, cold, numb of critical limb ischemia.
- For more proximal lesions, there will be claudicatio below the area of occlusion (ie, aortoiliac disease w claudication and decreased femoral pulses).
- **Acute ischemia:**
  - Most often caused by embolization from the hea commonly occur at bifurcations distal to the last mnemonic).
  - May also be 2° to cholesterol atheroembolism ("blu

ymphatic circulation that results in peripheral edema and
f the extremities. Primary (or congenital) lymphedema is
sed secondarily by surgeries involving lymph node dissec-
g countries, parasitic infections.

cause. Examples include:

patients present with unexplained swelling of the upper
dary to surgery).
ent with progressive swelling of the lower extremities bilat-
rdiac abnormalities (ie, filariasis infection).
with progressive, bilateral swelling of the extremities (1°).
rner syndrome will have lymphatic edema.

l. Rule out other causes of edema, such as cardiac and

ptom management, including exercise, massage therapy,
ments to mobilize and limit fluid accumulation.
ffective and relatively contraindicated.
ce for cellulitis with prompt gram-⊕ antibiotic coverage

y loss of consciousness and postural tone 2° to cerebral
logies can be cardiac, neurologic, or other.

- **Other:** Orthostatic BP readings (hypovolemia, autor
  glucose (hypoglycemia), and tilt-table testing (neurally

## Treatment

Tailored to the etiology.

Mediated Skin Disorders 50
NS 50
MA) 50
51
54
55
56
57
57
58
O ME/TOXIC EPIDERMAL NECROLYSIS 59
60
MPHIGUS VULGARIS 60
ifestations of the Skin 62
62
65
69
72
50 Ischemic Disorders of the Skin
DECUBITUS ULCERS
GANGRENE
Miscellaneous Skin Disorders
ACANTHOSIS NIGRICANS
LICHEN PLANUS
ROSACEA
PITYRIASIS ROSEA
VITILIGO
EYELID LESIONS
Neoplasms of the Skin
SEBORRHEIC KERATOSIS
ACTINIC KERATOSIS
CUTANEOUS SQUAMOUS CELL CARCINOMA
BASAL CELL CARCINOMA
MELANOMA
KAPOSI SARCOMA
MYCOSIS FUNGOIDES (CUTANEOUS T-C
CHERRY ANGIOMAS (HEMANGIOMAS)

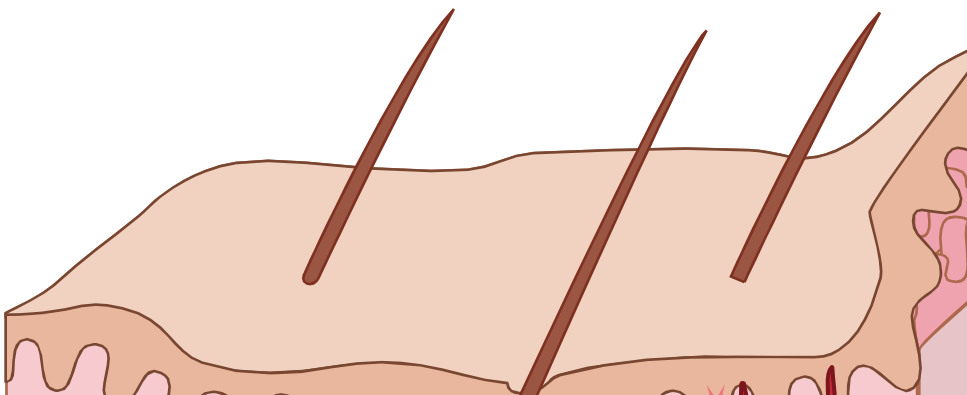
Table 2.2-2 outlines the types and mechanisms of hypersen

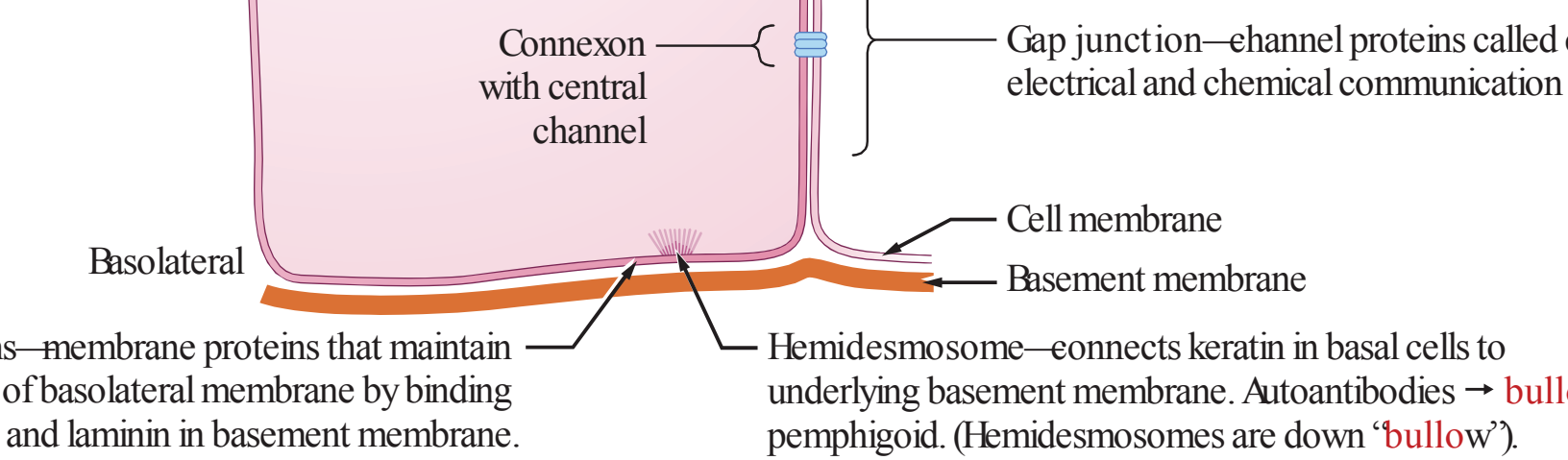
## ATOPIC DERMATITIS (ECZEMA)

A chronic inflammatory skin disorder that often starts in into adulthood. It is characterized by **pruritus leading to** Figure 2.2-3).

### History/Pe

- Look for a strong **family history** of **asthma, eczema, a** ("atopic triad").
- Patients are at ↑ risk of 2° bacterial (Staphylococcu (HSV or molluscum) infection due to constant waxing of pruritus and **scratching.**
- Triggers include climate, food, skin irritants, allergens, an
- Manifestations by age group:
  - **Infants:** Erythematous, edematous, weeping, pr plaques on the face, scalp, and extensor surfaces **The diaper area is often spared.**
  - **Children:** Dry, scaly, pruritic, excoriated papules, **ural** areas and neck.
  - **Adults:** Lichenification and dry, fissured skin in a f Often, there is hand or eyelid involvement.




Connexon with central channel
Gap junction—channel proteins called
electrical and chemical communication
Cell membrane
Basolateral
Basement membrane
s—membrane proteins that maintain
of basolateral membrane by binding
and laminin in basement membrane.
Hemidesmosome—connects keratin in basal cells to
underlying basement membrane. Autoantibodies → bull
pemphigoid. (Hemidesmosomes are down "bullow").

**helial cell junctions.** (Reproduced with permission from USMLE-Rx.com.)

can help distinguish eczema from a tinea infection. Mild IgE may be seen in some patients, but these are not used is.

**steroids are first-line therapy.** Topical immunomodula-nus) are useful as steroid sparing agents for moderate to r patients > 2 years of age.
should be used intermittently to avoid skin atrophy.
is to break the itching and scratching cycle. Use gentle urize after bathing, avoid irritants, and use antihistamines us.

Erythem
typically
and rese
red pap
with sur
halos. ↑
the pust
eruption
with no

## TIS

**nsitivity reaction** that results from contact with an aller-atient has previously been exposed and sensitized such as **perfumes/deodorants and neomycin.** More common in

| | |
|---|---|
| containing blister < 1 cm | Chickenpox (varicella (Figure E) |
| containing blister > 1 cm | Bullous pemphigoid |
| lined sac containing flui | Pilar cyst (epidermal |
| aining pus | Pustular psoriasis (Fig |
| nooth papule or plaque | Hives (urticarial) (Figu |
| of st atum corneum | Eczema, psoriasis, SC |
| dried serum, blood, pus | Impetigo (Figure J) |
| ression resulting from destruction of the epidermis and upper | Diabetic foot ulcer |
| y and thickening of the epidermis with accentuation of normal gs | Chronic scratching (p |



C D E

**instamine.** Reaction develops rapidly as a result of preformed antibody.

## t ype ii

**IgM** and **IgG** bind to antigen on an "enemy" cell, leading to lysis by complement or phagocytosis.




Cy-**2**-toxic.
Antibody and complement lead to MAC.

## t ype iii

Antigen-antibody complexes activate complement, which attracts PMNs; PMNs release lysosomal enzymes.

Imagine an immune complex as 3 things stuck together: antigen-antibody-complement.
Includes many glomerulonephritides and vasculitides.

Antibodies to the foreign proteins are produced in □5 days. Immune complexes form and are deposited in membranes, where they lead to tissue damage by fixing

Most serum sickness is now caused by drugs (not serum). Fever, urticaria, arthralgias, proteinuria, and lymphadenopathy occur 5–10 days after antigen exposure.

### Diagnosis

Clinical. **Patch testing** can be used to establish the cau
the acute-phase eruption has been treated.

### Treatment

First-line treatment is **topical corticosteroids and alle**
severe cases a systemic corticosteroid may be needed.

## SEBORRHEIC DERMATITIS

A common **chronic inflammatory skin disease** that m
hypersensitivity reaction to Malassezia furfur, a gener
found in **sebum and hair follicles.** It has a predilection
**ceous glands.hbbc**



nfused with atopic dermatitis, contact dermatitis, or psoria-

**elenium sulfide or zinc pyrithione shampoos** for the ntifungals (ketoconazole cream) and/or topical corticoste- Cradle cap often resolves with routine bathing and appli- in infants.

S

rmatitis due to **venous hypertension** forcing blood from perficial venous system. Venous hypertension is often a ve incompetence or flow obstruction. Commonly involves n patients with DVT history, chronic edema, and long If untreated, the area can become inflamed, exudative, d from hemosiderin deposition (see Figure 2.2-6). **Stasis .** Treat early with **leg elevation, compression stockings,** cal steroids.





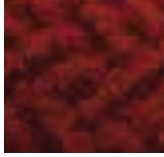


**FIGURE**
Shown a
vesicles
to areas
agent. (R
RM. Pathol
Correlation
1998.)



**FIGURE**
Venous
edema, a
permission

the nail plate, see Figure 2.2-7B).

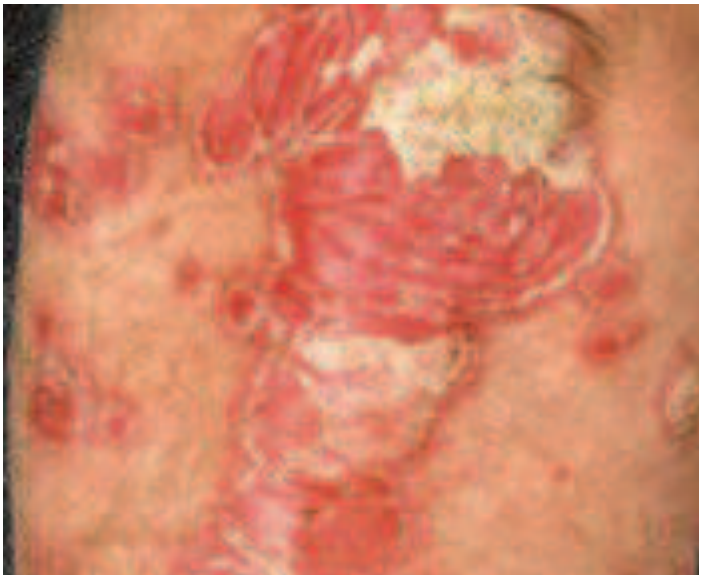
- Lesions initially appear small but may become confluent and provoked by local irritation or trauma **(Koebner phenomenon)**. Medications such as β-blockers, lithium, ACEIs, can worsen psoriasis.
- Up to 30% develop **psoriatic arthritis** (affecting small joints of hands and feet).

## Diagnosis

- Clinical. "Classical" presentation: **Auspitz sign (pinpoint bleeding when scale is scraped).**
- Biopsy if diagnosis is uncertain. Histology shows a thickened epidermis, elongated rete ridges, an absent granular cell layer, parakeratosis (nuclei in the stratum corneum), and a sterile neutrophilic microabscess in the stratum corneum **(Munro microabscess).**

## Treatment

- Local disease: Manage with **topical steroids,** calcipotriene (vitamin D derivative) and tazarotene (vitamin A derivative).
- Severe disease or presence of psoriatic arthritis: **Methotrexate, TNF biologics** (etanercept, infliximab, adalimumab) and **cyclosporine** can be used for extensive skin involvement (except in patients who can develop skin cancer from UV).





ome confluent and form circular or sinusoidal patterns.
c reactions, **extracutaneous manifestations** can include angioedema (deep, diffuse swelling often around the eyes Figure 2.2-8), asthma, GI symptoms, joint swelling, and

s a response to some often unidentified trigger: food, drug, , or physical stimulus (cold, heat, sun). Chronic urticaria hic.

It can often be difficult to determine the cause.

**systemic antihistamines.** Anaphylaxis (rare) requires epi-stamines, IV fluids, and airway maintenance.

morbilliform rash (most common, see Figure 2.2-9) to the ning toxic epidermal necrolysis (TEN). Maintain a high neous drug reaction in patients who are hospitalized and gs can cause **all four types of hypersensitivity reactions,** nay cause different types of reactions in different persons.





**FIGURE 2.2-9. Morbilliform rash.** Morbilliform rash following dru
duced with permission from Longo DL et al. Harrison's Principles of Internal Medicine, 18th ed. N
2011.)

### History/Pe

- Eruptions usually occur 7–14 days after exposure: **within 1–2 days of starting a new drug, it is probabl agent.**
- Eruptions are generally **widespread, relatively symmet**
- Most disappear within 1–2 weeks following remova agent.
- Extreme complications of drug eruptions include ery Johnson syndrome (SJS), and TEN.

### Diagnosis

Clinical impression. A skin biopsy may be helpful if the di

### Treatment

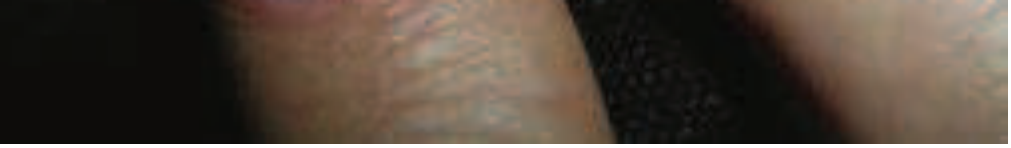
Discontinue the offending agent; treat symptoms with ant ical steroids to relieve pruritus.

## ERYTHEMA MULTIFORME

EM is a cutaneous reaction pattern with classic **targetoid** 2.2-10) that has many triggers and is often recurrent.

### History/Pe

- Typically, lesions start as erythematous macules tha



**ythema multiforme.** (Reproduced with permission from Dr. Richard Usatine.)

atment is all that is necessary; systemic corticosteroids are

n be treated with antipruritics; major cases should be

## SYNDROME/TOXIC EPIDERMAL NECROLYSIS

tute two different points on the spectrum of **life-threatening taneous diseases** that are often caused by a drug-induced on. **The epidermal separation of SJS involves < 10% of SA), whereas TEN involves > 30% of BSA.**

evere mucosal erosions with widespread erythematous, puric macules or atypical targetoid lesions (see Figure 2.2- nal lesions often become confluent and show a ⊕ **Nikol-**



- Include staphylococcal scalded-skin syndrome (SSSS tion (usually after bone marrow transplant), radiation on the differential diagnosis.

### Treatment

- High risk of mortality. **Early diagnosis and discontin agent are critical** in improving survival.
- Patients have the **same complications as burn victims** and electrolyte disturbances and 2° infections, so cove age fluids and electrolytes.
- Data on pharmacologic therapy with steroids, cyclos mixed.

## ERYTHEMA NODOSUM

A **panniculitis** (inflammatory process of the **subcutane** triggered by infection (Streptococcus, Coccidioides, Yersi tions (sulfonamides, antibiotics, OCPs), and chronic in (sarcoidosis, Crohn disease, ulcerative colitis, Behçet disea

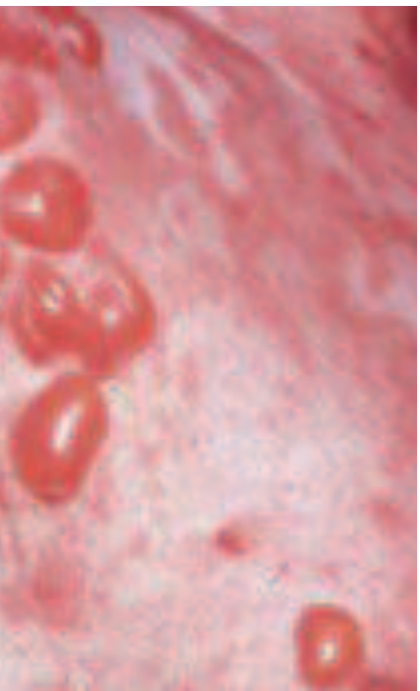
### History/Pe

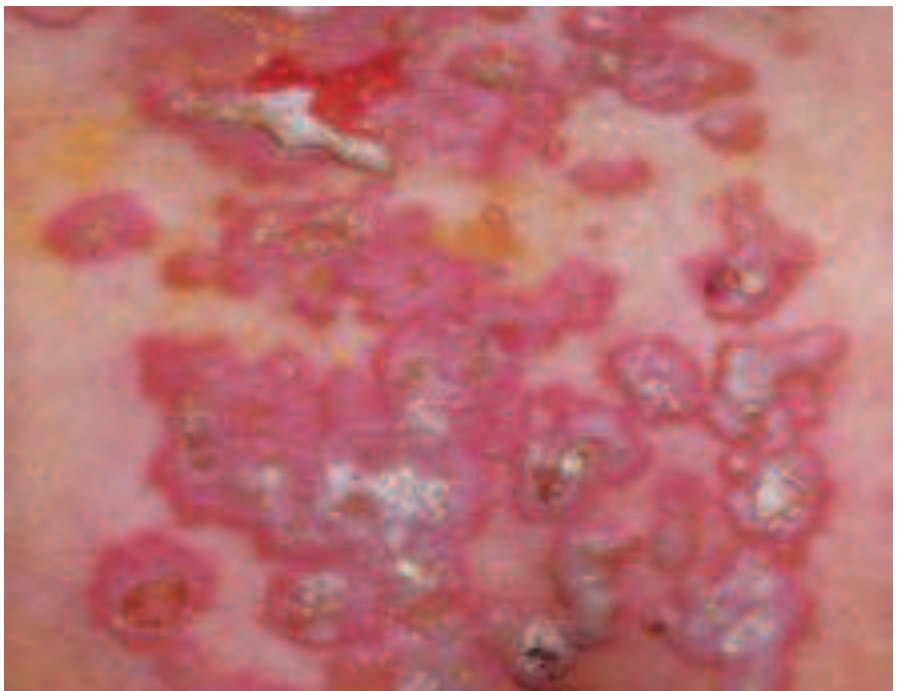
- **Painful, erythematous nodules** appear on the patie (see Figure 2.2-12) and slowly spread, turning brown c present with fever and joint pain.
- Patients with erythema nodosum may have a false-⊕ V

### Diagnosis

Clinical. Workup with an ASO titer, PPD in high-risk pat out sarcoidosis, or inflammatory bowel disease workup ba complaints.

| ⊖ | ⊕ |
| --- | --- |
| Rare. | Common. |
| Usually > 60 years of age. | Usually 40–60 years of ag |
| Generally idiopathic. | ACEIs, penicillamine, phe |
| Rare and milder course. | Possible. |
| Clinical. Most accurate test: **skin biopsy with direct immunofluo escence,** and/or ELISA. | Same as that for bullous |
| Steroids (prednisone). | High-dose steroids (pred therapy (azathioprine, m rituximab). |





causes erythema and swelling.

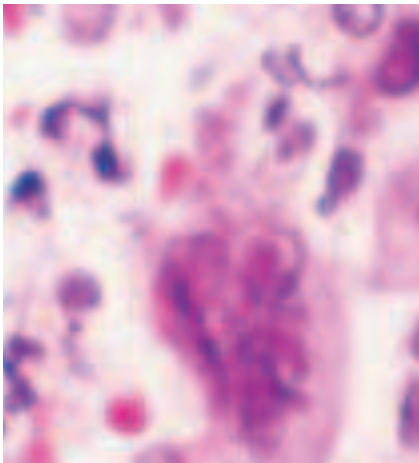
### History/Pe

- The initial infection is by direct contact, after wh
  remains dormant in local nerve ganglia. **1° episodes a**
  **and more severe** than recurrences.
- Onset is preceded by prodromal tingling, burning, o
  present with lymphadenopathy, fever, discomfort, ma
  involved tissue.
- Recurrences are limited to mucocutaneous areas
  involved nerve.
  - **Recurrent oral herpes (HSV-1):** The common **"c**
    labialis, which presents as a cluster of crusted vesic
    tous base (see Figure 2.2-15A). Often triggered by s
  - **Recurrent genital herpes (HSV-2):** Unilateral and
    cluster of blisters on an erythematous base, but wi
    temic involvement than the 1° infection.

### Diagnosis

- Clinical. **Viral culture of lesion** is the most accurate
  cent antigen is the most rapid.
- Classic multinucleated **giant cells on Tzanck smea**
  support the diagnosis.

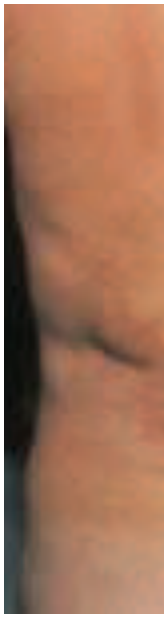




**recurrences (more than six outbreaks per year).** Daily
acyclovir, famciclovir, or valacyclovir.
s, HSV can persist, with ulcers remaining resistant to anti-
mptomatic HSV infection lasting > 1 month can be con-
-defining illness.

## us (VZV)

ferent diseases, varicella and herpes zoster—with transmis-
espiratory droplet or by direct contact. VZV has an incu-
–20 days, with contagion beginning 24 hours before the
d lasting until lesions have crusted.



of malaise, fever, headache, and myalgia occurs 24 hours
h.
ns appear in crops over 2–3 days, evolving from red mac-
es that then crust over.
time, patients may have **all stages of lesions present.** The
calp, and mucous membranes are involved.
ickenpox is often more severe, with **systemic complica-**
**pneumonia** and **encephalitis.**

**(shingles)** represents the recurrence of VZV in a specific
esions cropping up along the nerve's **dermatomal** distribu-
ks are usually preceded by intense local pain followed by
ers on an erythematous base (see Figure 2.2-16). Zoster
disseminated in immunocompromised persons.
ts with zoster can develop **postherpetic neuralgia** (severe
at persists for months at the infection site).

**FIGURE**
The uni
of the gr
tous base
permission
Synopsis of
McGraw-H

Immuno
patients
chemot
severely



**conta-**
Richard

- **Lesions are asymptomatic** unless they become inflam

### Diagnosis

Clinical. If the diagnosis is uncertain, most accurate tes
**large inclusion or molluscum bodies** seen under the mic

### Treatment

- Local destruction: curetting, freezing, or applying tri
the lesions.
- In kids, lesions resolve spontaneously over months to
left untreated.

## Verrucae (Warts)

Warts are caused by **HPV** and can occur on skin, muco
other epithelia. Although usually benign, **some subtypes
16 and 18)** lead to **squamous malignancies**. Spread is by

### History/Pe

- Common warts are most often seen on the hands, tho
anywhere.
- Classic genital warts (condyloma acuminatum, caused
and 11) are **cauliflower-like papules or plaques** appe
the vulva, or the perianal region (see Figure 2.2-18).
- Mothers with genital HPV can transmit laryngeal wa
aspiration during delivery.





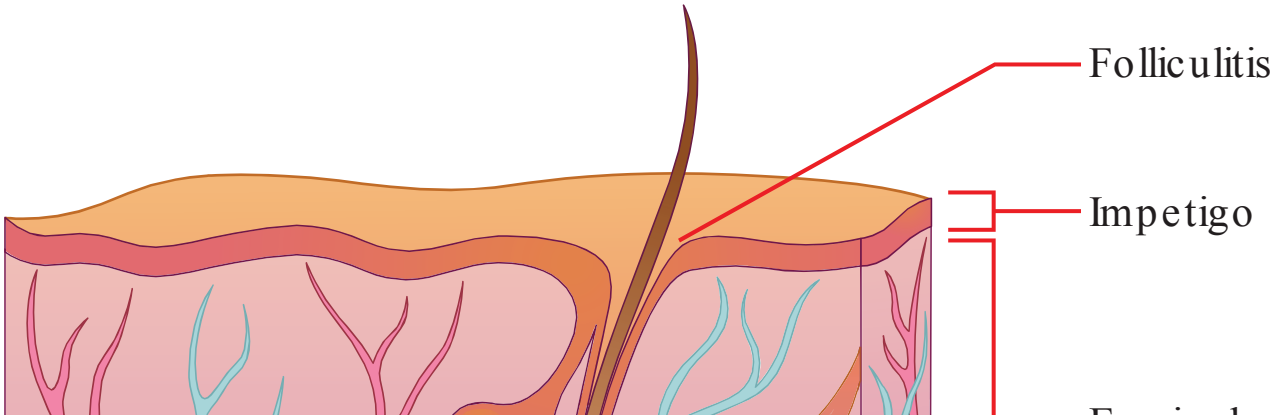
the **epidermis** that primarily occurs in children and is **up A streptococcal and staphylococcal** organisms. It is ct contact. **Streptococcal impetigo can be complicated cal glomerulonephritis.**

Pustules and **honey-colored crusts** on an erythematous e face around the mouth, nose, or ears (see Figure 2.2-20). haracterized by bulla in addition to crusts that can involve s. **Bullous impetigo is almost always caused by** S aureus nto SSSS.



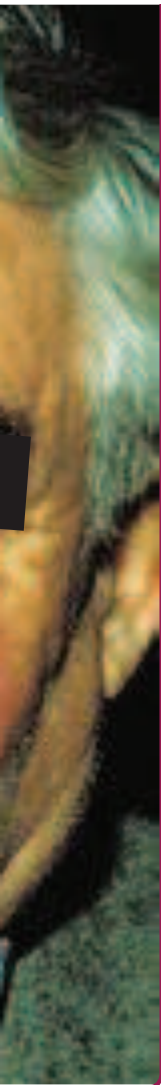
**FIGURE**
pustules
crust are
the nose
sion from E
apy, 1st ed

antistaphylococcal activity based on severity and MRSA



eat
hird-

or



of the
l. Fitzpat-
d. New

litis

increasingly common cause of purulent cellulitis. Risk fa
tes, IV drug use, venous stasis, and immune compromise.

### History/Pe

- Presents with **red, hot, swollen, tender skin.** Fever
  mon.
- **Erysipelas** is a type of cellulitis usually caused by s
  confined to the dermis and lymphatic tissue, creating
  **raised, indurated, well-demarcated, erythematous** ar
  ure 2.2-21).

### Diagnosis

- Clinical. Cultures may aid in diagnosis and help deter
  sitivities for treatment if pus or sepsis is present.
- Rule out abscess, osteomyelitis, and necrotizing fasciiti

### Treatment

- Topical antibiotics are ineffective due to depth of infec
- Use 5–10 days of oral antibiotics. IV antibiotics are
  dence of systemic toxicity, comorbid conditions, diab
  extremes of age, or hand or orbital involvement. Antib
  to impetigo.

## Necrotizing Fasciitis

Deep infection along a **fascial plane** causing severe pain
sia. It is usually caused by a **mixed infection of anaerob
teria that includes** S aureus, Escherichia coli, **and** Clost
Ten percent of cases are caused by S pyogenes. A history
surgery to the affected area is sometimes elicited.

### History/Pe

- Acute onset of pain and swelling progressing to anes



**crotizing fasciitis of the lower extremity.** Patient presented with necrotizing fasciitis and myositis due to β-hemolytic streptococcal
permission from Brunicardi FC et al. Schwartz's Principles of Surgery, 9th ed. New York, NY:

f necrotizing fasciitis based on clinical exam and imag- soft tissue) requires **immediate surgical exploration and** culture helps determine the organisms involved.

**gency:** Early and aggressive surgical debridement is criti-

**broad-spectrum coverage** is necessary. If Streptococcus organism involved, penicillin G is the drug of choice. second line and also used to decrease toxin production by aerobic coverage, give metronidazole or a third-generation

r infection of a hair follicle. Typically caused by infection s, Streptococcus, and gram-⊖ bacteria. Occasionally can uch as Candida albicans or Malassezia furfur. Can occur **ith follicles.**

y pustule at the opening of a hair follicle with a hair pene- the infection is deeper, a **furuncle,** or hair follicle abscess,

Fournier
necrotiz
the gen

Pseudon
to "hot t

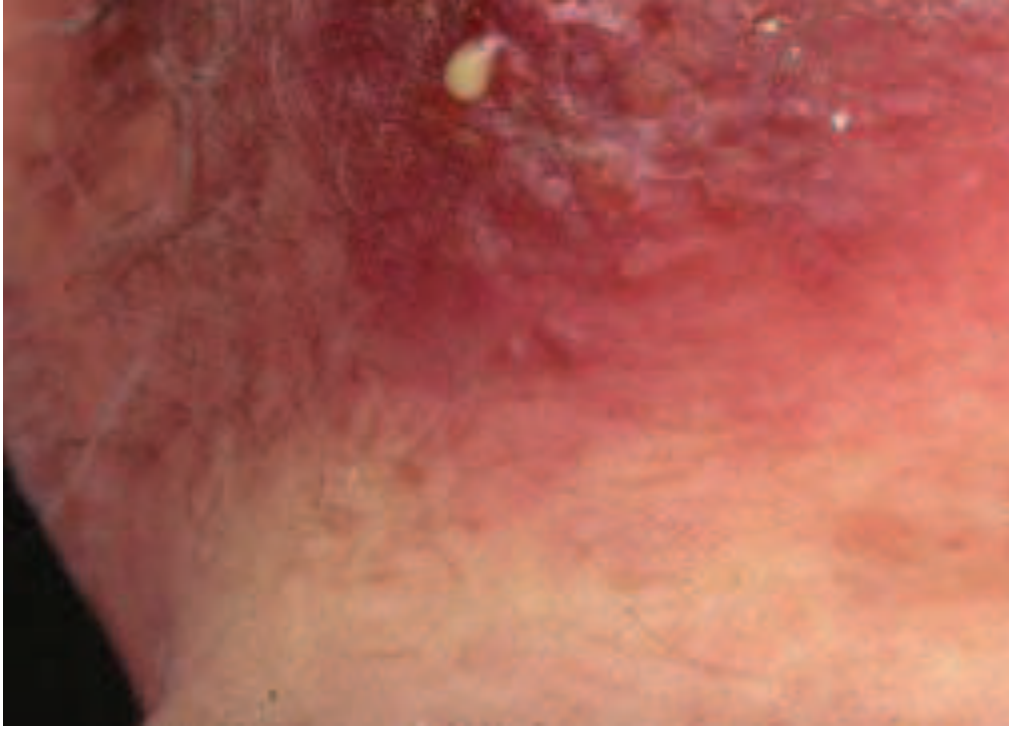


**FIGURE 2.2-23. Carbuncle due to methicillin-sensitive** S aureus. matory plaque studded with pustules, draining pus, on the nape of the down to the fascia and has formed from a confluence of many furuncle sion from Wolff Ket al. Fitzpatrick's Color Atlas &Synopsis of Clinical Dermatology, 7th ed. New

### Tr ea Tmen T

Topical antibiotics (mupirocin) treat mild superficial d disease is treated similarly to impetigo, with cephalexin escalating to clindamycin if MRSA is suspected.

## Acne Vulgaris

A skin disease common among adolescents. The pathog **monal activation of sebaceous glands,** the developm **(plugged pilosebaceous follicle),** and involvement of acnes **in the follicle,** causing inflammation. Acne lesion medications (lithium, corticosteroids) or by topical occlus

### His To r y/Pe

- There are three stages of acne lesions:
  - **Comedonal:** Open (**"blackheads"**) or closed (**"wh**

e failing, **oral retinoids (isotretinoin)** are the most effec-
ll other medications are stopped.
s a **teratogen** and **elevates LFTs.** Patients require **periodic**
**o check LFTs, cholesterol, and triglycerides.**

**crococcygeal region.** Not all contain hair or are true cysts.
ought to start as a folliculitis that becomes an abscess com-
microbes, especially Bacteroides, as a result of repetitive
ion in 20- to 40-year-old men.

bscess at the natal cleft that can be tender, fluctuant, and
s associated with purulent drainage or cellulitis. Systemic
common, but cysts can develop into perianal fistulas.
ude deep and hairy natal clefts, obesity, and a sedentary

**ion and drainage** of the abscess followed by sterile pack-
.
ene and shaving of the sacrococcygeal skin can help pre-

NS

zia furfur, a yeast that is part of normal skin flora. Humid

Antibiot
cysts un
antibiot
and ana





B

**color. (A)** Note the discrete, hypopigmented patches extensively involving the patien
gmented macules or patches in some individuals. **(B)** KOH preparation shows the ch
duced with permission from Tintinalli JE et al. Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 7th
om Wolff K et al. Fitzpatrick's Dermatology in General Medicine, 7th ed. New York, NY: McGraw-Hill; 2008.)

### Treatment

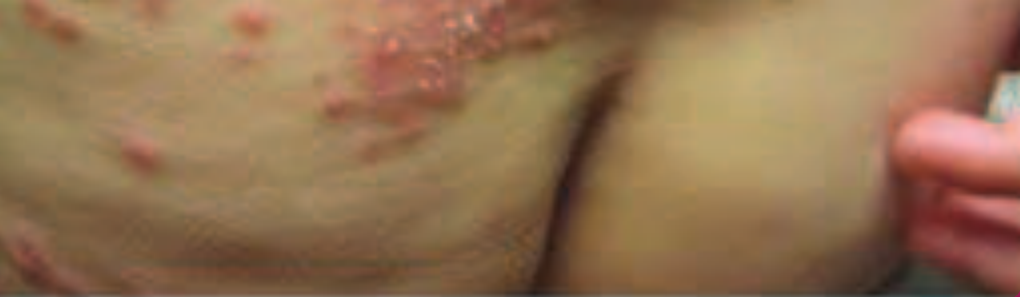
Treat lesions with topical **ketoconazole or selenium sulfi**

## Candidiasis

"Yeast infection" or "thrush," candidiasis can be caused b
cies but is most commonly caused by C albicans. In
patients, it typically presents as a superficial infection of
membranes **in moist areas such as the groin, skin folds,
and below the breasts.** Oral thrush is common in infanc
often **a sign of a weakened immune system.**

### History/Pe

- Patients often have **history of antibiotic or steroid us
nocompromise.**
- **Oral candidiasis:** Presents with painless white plaque
**scraped off.**
- **Candidiasis of the skin:** Presents as markedly erythem
occasional erosions and **smaller satellite lesions** (see
nearby, often in skin folds. In infants, infection is ofte
area and along the inguinal folds.



**taneous candidiasis with satellite lesions.** (Reproduced with permission from Dermatology in General Medicine, 8th ed. New York, NY: McGraw-Hill; 2012.)

**n) candidiasis:** Topical antifungals; keep skin clean and

pical nystatin.

## ctions

y live in **tissues with keratin (skin, nails, and hair)** and se of infection. **Causative organisms include** Trichophy-**),** Microsporum, **and** Epidermophyton species. Risk fac-es, ↓ peripheral circulation, immune compromise, and of skin (from athletic activities).

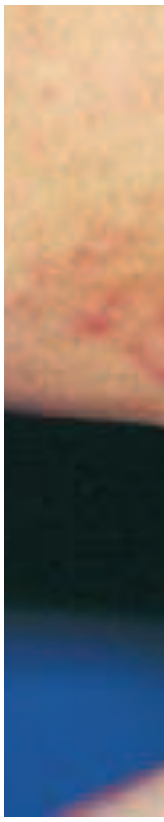
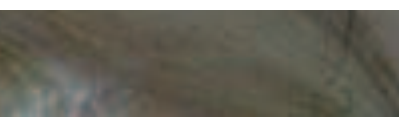


**FIGURE**
Note the
scaly, er
central c
from Wolff
Medicine, 7

subtype:

Scaly, pruritic eruption with a sharp, irregular border, al clearing (see Figure 2.2-26). Can be seen in immuno-tients or in children following contact with infected pets.

**nuum:** (see Figure 2.2-27A): Presents as chronic interdigi-erosions between the toes **("athlete's foot")** or as a **thick-on the soles** (moccasin distribution). In addition, involve-d is typical in the "one hand two feet syndrome."







### Treatment

**Start with topical antifungals;** escalate to oral if infecti
unresponsive to topicals. **Tinea capitis must be treated**
**tions** to penetrate into hair follicles; oral treatment should
in immunocompromised patients.

## PARASITIC INFECTIONS

### Lice

Lice live off blood and on specific parts of the body, depe
cies (head lice, body lice, pubic lice). Lice are spread th
or by the sharing of bedclothes and other garments or ha
secrete local toxins that lead to pruritus.

#### History/Pe

- Patients with lice often experience severe pruritus, an
  tion of the excoriations is a risk. **Classroom epidem**
  common.
- Body lice are seen in people with inadequate hygie
  crowded living conditions. Pubic lice (called "crab
  squat, crablike body shape) contain anticoagulant in
  bites often turn blue.

#### Diagnosis

Lice and their eggs (nits) can be seen on hairs or in clot
**eye.**

#### Treatment

- **Head lice:** Treat with topical permethrin, pyrethrin,
  mechanical removal.

be treated with 5% **permethrin from the neck down infants),** and their contacts should be treated as well. Oral o effective.
ist up to 2 weeks after treatment.
ding should be thoroughly washed as for lice.

## Disorders of the Skin

S

ic necrosis following **continuous pressure on an area of** crocirculation.

ly seen in **bedridden patients** who lie in the same spot erlying bony prominence or lack of fat ↑ the likelihood of **crum, heels).** Patients lacking mobility or cutaneous sen- isk. Incontinence of urine or stool may macerate the skin, n.

**ey:** Routinely move bedridden patients; special beds can re.
ops, low-grade lesions can be treated with routine wound ydrocolloid dressings. High-grade lesions require **surgical**

Lice can
eye. Sca
to be id
dermato

Q

A 42-y
cellulit
swimm
well-d
the an
Antibi
the pa
erythe
aroun
aspect
hue. W

injury. **A medical emergency.**

### Diagnosis

Clinical. Air in soft tissue on x-ray film is very suggestive (s

### Treatment

- Surgical **debridement,** with amputation if necessary treatment. **Antibiotics alone do not suffice** by virtue flow, but they should be given as an adjuvant to surgery
- Hyperbaric oxygen (toxic to the anaerobic C perfringen **debridement** to help with treatment.

## Miscellaneous Skin Disorders

### ACANTHOSIS NIGRICANS

- A condition in which the skin in the **intertriginous** genitals, axillae) is hyperkeratotic and **hyperpigmen appearance** (see Figure 2.2-29).



ion.
s with **violaceous, flat-topped, polygonal papules** (see
**Wickham striae (lacy white lines) may be present on**
ons may demonstrate prominent **Koebner phenomena**
ear at the site of trauma).
re treated with **topical corticosteroids.** For severe disease,
teroids and phototherapy may be used.

er of pilosebaceous units whose etiology is unclear. Early
**entral facial erythema** is seen with **telangiectasias.** Later,
tules may develop. Longstanding facial rosacea may lead
wth of nasal connective tissue known as **rhinophyma** (see
Ocular rosacea can predispose to blepharitis, stye and cha-
.



**FIGURE**
**cans.** Ve
thickeni
promine
(Reproduce
RA, Saaved
Clinical Der
Hill; 2013.)

Lichen p
**P**ruritic,
**P**apules

soles,

An acute dermatitis of unknown etiology that has been h
resent a reaction to a **viral infection with human he**
because it tends to occur in mini-epidemics among young

- **Hx/PE:**
  - Initial lesion is classically a **herald patch** that is e
    peripheral scale.
  - Days to weeks later, a 2° eruption appears: multi
    and plaques with a fine **"cigarette paper"** scale
    Papules are arranged along skin lines, giving a
    **tree" pattern** on the patient's back.
- **Dx: Clinical.** Confirm with KOH exam to rule out
  patch may be mistaken for tinea corporis). Consider t
  syphilis, which can present similarly.
- **Tx:** Rash **heals in 6–8 weeks without any treatment.**
  to manage symptoms includes skin lubrication, antipru
  mines.

## VITILIGO

**Acquired loss of function or absence of melanocytes** lea
of depigmentation. Probably 2° to autoimmune disease.



oft and **yellow** plaques seen on the medial aspects of the
y, associated with hyperlipidemia and 1° biliary cirrhosis.
**inful** acute eyelid gland infection (stye), usually due to
ated on the edge of the lid.
onic inflammatory **painless** cyst due to a blocked eyelid

## ns of the Skin

### TOSIS

**n tumor that appears in almost all people after age 40.**
known. Lesions have no malignant potential (see Figure

ytic, waxy brown papules and plaques with superficial ker-
igure 2.2-34). Lesions may appear in great numbers and
**n" appearance.**
ome irritated either spontaneously or by external trauma.

nay appear similar to melanoma, a biopsy may be prudent

excision or curettage.

**hands.** (
Usatine.)



**FIGURE**
**sis.** (Used

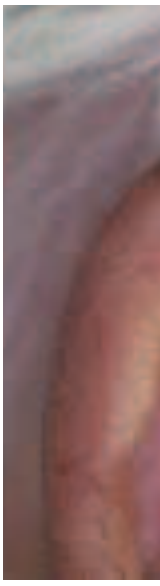


**FIGURE**
**sis.** The
erythem
scale. (Re
RM. Pathol



**cell car-**
ceration
t lesions
n or
n from
nical-
CT: Apple-

The second most common skin cancer, with locally destru
as the potential for **metastasis and death. Sun exposur**
mon causative factor, but exposure to **chemical carcinog**
therapy, sites of **burns or chronic trauma** (as in draining
osteomyelitis), and chronic immunosuppression (as in tr
also predispose patients to developing SCC.

### History/Pe

- SCCs have many forms, and a single patient will often ha
- Most SCCs occur in older adults with sun-damaged
  actinic keratoses, presenting as erythematous, **ulcerate**
  (see Figure 2.2-36).
- **Marjolin's ulcer** is a type of rare SCC that arises in sit
  ulcers.
- **Arsenic exposure** is a rare cause of multiple SCCs in
  **tribution.**
- SCCs from actinic keratoses rarely metastasize, but the
  and ulcers are more likely to do so. SCC occurs on
  commonly than basal cell carcinoma.

### Diagnosis

Clinical. Confirm with shave biopsy.

### Treatment

Surgical excision or **Mohs surgery** (very thin slices are ex
with a microscope via frozen section). Lesions with high
may need radiation or chemotherapy.

## BASAL CELL CARCINOMA

The most common malignant skin cancer, BCC is slow
destructive but has **virtually no metastatic potential. Cu**

also ↑ risk. Some patients inherit a predisposition to mel-
**ilial atypical mole and melanoma (FAM-M) syndrome.**
btypes (see Table 2.2-4).

nomas begin in the epidermal basal layer, where melano-

nomas may metastasize **anywhere in the body** (lung, liver,
hree to 5% of patients with metastatic melanoma have no

n and treatment are essential. Screening exams using the
, and dermoscopy may detect melanoma early when it is
ure 2.2-38). An **excisional biopsy** should be performed on
sion. **Malignancy is determined histologically.**
anging skin lesion is a suspicious feature for malignant

nomas are staged by Breslow thickness (depth of invasion
nd by tumor-node-metastasis **(TNM) staging.** Ulceration
tic sign.

**of Melanoma**

| pre Sentation |
|---|
| f all melanomas. Can occur at any age but is seen in young adults. presents on the trunk in men and on the legs in women. A prolonged ntal growth allows for early diagnosis, when it is still confined o the mis. |
| s have a rapid vertical growth phase and appear as a fast-growing |

**FIGURE**
**carcinom**
telangiec
from Wolff
Clinical Der
Hill; 2005.)

The AB

**A**symme
Irregular
Variation
**D**iamete
**E**volution

. **(A)** Superficial spreading; **(B)** nodular; **(C)** lentigo; **(D)** acral. (Used with permission from

### Treatment

- Lesions confined to the skin are treated by **excision w**
  nel lymph node biopsy is useful for staging but does
  motherapy, biologic and radiation therapy may be u
  metastatic melanoma.
- Patients with early melanoma are at low risk for recurr
  risk for the development of **subsequent melanomas. M**
  **anomas may recur or metastasize at a higher rate. Pa**
  thus essential.

## KAPOSI SARCOMA

A vascular proliferative disease that has been attribute
HHV-8, which is also called Kaposi sarcoma–associated h

### History/Pe

- Presents with multiple red to violaceous macules, papu
  can progress to plaques on the lower limbs, back, face,
  lia (see Figure 2.2-39).
- Plaques can also be found in the GI tract and lung.
- **HIV-associated (epidemic) KS** is an aggressive form
  although less common since the advent of HAART,
  **common HIV-associated malignancy.**

### Diagnosis

Diagnosed by history, clinical impression, and histology.

### Treatment

Start HAART therapy if patient is HIV positive. Small l

inical features and histology, with immunologic charac-
ectron microscopy showing the typical **Sézary or Lutzner**
**m lymphocytes).**
clinically indistinguishable from dermatitis, **so histologic**
**cated for any dermatitis that is chronic and resistant to**

mainstay of treatment for many patients. Early localized
to total skin electron beam irradiation. For more exten-
sease, radiation therapy is an effective option. Treatment
g steroids, chemotherapy, retinoids, monoclonal antibod-
en combined.

## (HEMANGIOMAS)

d papules that can appear anywhere on the body (see
the most common **benign** vascular tumor, and it often
No treatment necessary, but can be excised for cosmetic



**FIGURE**
**des.** Ma
face lead
with permi
matology i
McGraw-H



**FIGURE**
**gioma.** (
Usatine.)

Metabolism
S
S
TIo N
To XIc o SIS
rders

84 Pituitary and Hypothalamic Diso
84 HYPo PITUITARISM
84 c USHING SYNd Ro ME
88 Ac Ro MEGALY
89 HYPERPRo LAc TINEMIA
89 d IABETES INSIPId US
89 SIAd H
93 Adrenal Gland Disorders
94 Ad RENAL INSUFFIc IENc Y
94 PHEo c HRo Mo c YTo MA
95 HYPERALd o STERo NISM
96 c o NGENITAL Ad RENAL HYPERPLASIA
97 Multiple Endocrine Neoplasias
99

unexplained weight loss. Patients may also present wi
Table 2.3-2).

- Usually affects nonobese children or young adults.
- Associated with HLA-DR3 and -DR4.
- Type I DM diagnosed as an adult is known as latent au
of adults (LADA).

### Diagnosis

- At disease onset, **anti–islet cell, anti–glutamic acid d
GAD) antibodies, anti-insulin or anti-Zn transporter**
present in serum.
- At least one of the following is required to make the dia
  - A random plasma glucose level ≥ 200 mg/dL plus sy
  - A fasting (> 8-hour) plasma glucose level ≥ 126 mg
prandial glucose level ≥ 200 mg/dL following an or
test. (Requires repeat testing to confirm unless unec
mia.)
  - Hemoglobin $A_{1c}$ > 6.5%. (Requires repeat testing
unequivocal hyperglycemia.)

### Treatment

- Insulin injections (see Table 2.3-1 and Figure 2.3-1)
glucose in the normal range (80–130 mg/dL preprandi
dL postprandial levels). **Encourage routine hemoglob
3 months.** Goal hemoglobin $A_{1c}$ < 7% (< 7.5% in chil
glucose and **hemoglobin $A_{1c}$** levels can be tolerated
very young and the very old, or those with multiple m
light of the ↑ risk of hypoglycemia.
- Consider the use of an insulin pump, which provides
acting insulin infusion.
- Table 2.3-1 outlines general health maintenance guide

| | Mechanism | Side effects | |
|---|---|---|---|
| | Inhibits hepatic gluconeogenesis and ↑ peripheral sensitivity to insulin. | Weight loss, GI upset, and rarely, lactic acidosis. | C<br>(<br>**i**<br>h |
| e,<br>) | ↑ endogenous insulin secretion. | **Hypoglycemia** and **weight gain.** | |
| zone)[a] | ↑ insulin sensitivity. | Weight gain, **edema,** hepatotoxicity, and bone loss. | C<br>**v** |
| rs | ↓ intestinal absorption of carbohydrates. | Flatulence and hypoglycemia. | |
| **liptin,**<br>**-liptins**) | Inhibit the degradation of GLP-1. | **Weight neutral.** | |
| **-tides**) | GLP-1 agonists. Delay absorption of food; ↑ insulin secretion and ↓ glucagon secretion. | Injected subcutaneously (weekly exenatide now available). Nausea and, rarely, pancreatitis. Can cause **weight loss.** | |
| ner | Inhibit SGLT2 in proximal tubule to ↓ glucose reabsorption. | **UTIs,** vulvovaginal candidiasis. Can cause **weight loss** and ↓ blood pressure. | |
| | Given alone or in conjunction with oral agents. Types of insulin include regular, short-acting | Weight gain and hypoglycemia. | |

Strict BP control to < 140/80 mm Hg; ACEIs/ARBs are firs -line agents.

Annual physical examination to screen for cardiovascular disease (BP and lipid mon

(test for microalbuminuria), retinopathy (dilated-eye exams), and neuropathy (foot

All diabetic patients > 19 years should receive the pneumonia vaccine.

and Drug Administration restricted access to rosiglitazone because of concern for ↑ car
ted to patients currently on the medication who acknowledge that they understand the
ycemic control with other medications.

ofessional/StatementsGuidelines/Prevention-Guidelines_UCM_457698_SubHomePage

- **Nonketotic hyperosmolar hyperglycemia** may be se
  poor glycemic control (see Table 2.3-2).
- Usually occurs in older adults with obesity (often ab
  strong genetic predisposition; diagnosed increasingly in
- Risk factors include obesity, rapid weight gain, a ⊕ f
  entary lifestyle, increasing age, and other componen
  drome (see below).

Diagnosis

- Diagnostic criteria are the **same as those for type 1 DM**
- Anti–islet cell and anti-GAD antibodies will be ⊖.

ispro, glulisine

ular




Detemir

| | |
|---|---|
| **acetone breath odor.** | |
| Glucose > 250 mg/dL.<br>**Metabolic acidosis** (bicarbonate < 18 mEq/L).<br>↑ **urine and serum ketones.**<br>↑ **anion gap.**<br>Serum osmolality **normal.** | Glucose > **600 mg/dL.**<br>**No acidosis** (bicarbona<br>No ketones.<br>**Normal anion gap.**<br>**Serum osmolality > 32** |
| Fluids, potassium, continuous **insulin (caution: will lower potassium),** phosphorus. Bicarbonate rarely used (only if pH is < 6.9). Treat initiating event. Monitor response to treatment by **closure of the anion gap.** | **Aggressive fluid ,** elec<br>Treat the initiating even |

**nmendations:**

**eening all patients with risk factors for diabetes (hyper-sity, family history, racial/ethnic minorities).**

**no risk factors:** Test hemoglobin $A_{1c}$ at age 45; retest every oglobin $A_{1c}$ is < 5.7% and no other risk factors develop.

**nic Complications of DM**

| | **d**eScription |
|---|---|
| Appears when diabetes has been present for **at least 3–5 years** (see Figure 2.3-2). Prev control of hyperglycemia and hypertension, annual eye exams, and **laser photocoagu neovascularization.** | |
| Characterized by **glomerular hyperfilt ation** followed by **microalbuminuria.** Seen in **years.** Preventive measures include **ACEIs** or ARBs and BP/glucose control. Kimmelstie | |

A B

**FIGURE 2.3-2. Diabetic retinopathy. (A) Nonproliferative retino** dates, dot-blot hemorrhages, and microaneurysms. **(B) Proliferative re** macular edema, vitreous traction, and neovascularization of the retinal with permission from USMLE-Rx.com.)

- **Patients with impaired fasting glucose (> 100 mg dL) or impaired glucose tolerance:** Follow up with

### Treatment

See Table 2.3-1 for treatment options. Treat to goal he < 7% (corresponds to an average glucose of 154 mg/dL goals in the elderly who are at risk of hypoglycemia.

### Complications

See Tables 2.3-2 and 2.3-3 for an outline of the complicat

## METABOLIC SYNDROME

Also known as insulin resistance syndrome or syndrome X ↑ risk of CAD and mortality from a cardiovascular event.

### History/Pe

Presents with abdominal obesity, high BP, impaired gly dyslipidemia.

### Diagnosis

**Three out of five** of the following criteria must be met:

- Abdominal obesity (↑ waist girth): ≥ 40 inches (102 cm inches (88 cm) in women.

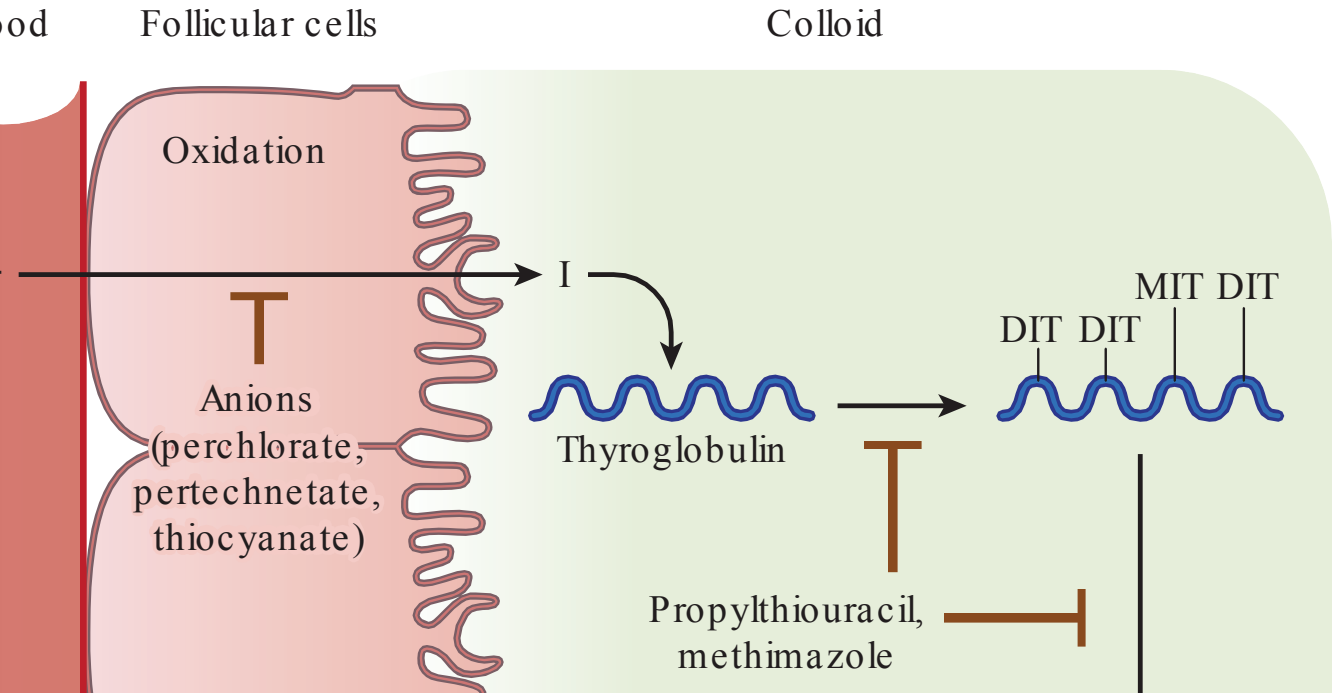
**assessment of thyroid function,** unless there is a history
g, tumor, radiation, trauma), in which case free $T_4$ should

**ine uptake (RAI) and scan:** Shows the level and distribu-
take by the thyroid. Determines if a nodule is functioning
yperthyroidism, or nonfunctioning and requires a biopsy
vorkup. See Table 2.3-5.
**rement:** Not an adequate screening test. Ninety-nine per-
g $T_4$ is bound to thyroxine-binding globulin (TBG). Total
altered by changes in levels of binding proteins.
**ement:** The **preferred screening test for thyroid hormone**

## M/THYROTOXICOSIS

a state of ↑ **synthesis** of $T_3/T_4$; thyrotoxicosis is a state of
ost commonly due to **Graves disease,** but can also result
ee Table 2.3-4).

The **autoimmune** form of hyperthyroidism. **Thyroid-**
**bodies** ↑ synthesis of $T_3/T_4$.




↑ TBG ca
estroge
You do

Exophth
and thy
disease.

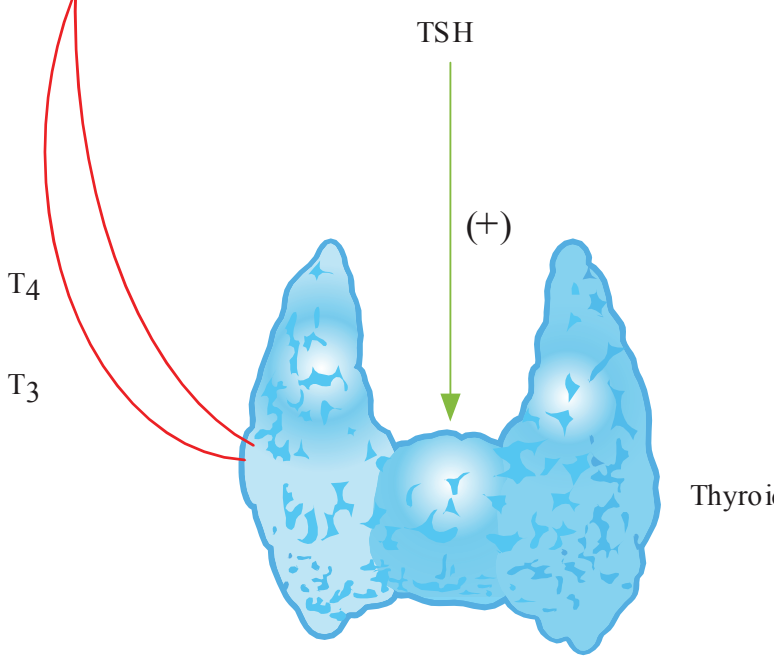



**FIGURE 2.3-4. The hypothalamic-pituitary-thyroid axis.** (Reproduc
Molina PE. Endocrine Physiology, 3rd ed. New York, NY: McGraw-Hill; 2009.)

dies are
sease.

- **Toxic adenoma/toxic multinodular goiter:** Result in h to autonomous hyperactive thyroid nodules.
- **Thyroiditis:** Due to transient inflammation of the release of previously synthesized thyroid hormone; ↑ in circulating $T_3/T_4$. A hypothyroid phase may follo phase with eventual return to normal in many.
- **Fetal thyrotoxicosis:** Classically in an infant born Graves. TSH-stimulating antibodies are IgG and can c

**d Disease**

| t Sh | $t_4$ | $t_3$ | CauSeS |
|---|---|---|---|
| ↓ | ↑ | ↑ | Graves disease, toxic multinodular goiter, toxic adenoma, a postpartum thyrotoxicosis, postviral thyroiditis. |
| Nl/↑ | ↑ | ↑ | Rare. Caused by TSH-producing pituitary adenoma. TSH of **normal** (not suppressed). |

| | | | |
|---|---|---|---|
| | | uptake | |
| lar | ↓ | Low uptake | ↑ |
| | ↓ | Low uptake | ↓ |

**ight loss, heat intolerance, anxiety, palpitations, ↑ bowel pathy/proximal muscle weakness,** insomnia, and men- ties.

eals warm, moist skin, goiter, sinus **tachycardia,** or **atrial** , fine **tremor, lid lag,** and hyperactive reflexes. **Exoph- stimulation of orbital fibroblasts by antibodies),** pre- , and thyroid bruits are seen only in Graves disease (see

hoice is a serum TSH level, followed by $T_4$ levels and, SH is low and free $T_4$ is not elevated). May need addi- rovided by RAI scans (see Figure 2.3-6) and thyroglobulin 3-4 and 2.3-5.

**eatment: Propranolol (or metoprolol, atenolol)** to man- mptoms.

**treatment:** Antithyroid drugs (methimazole or propylthio-

Thyroid threaten that ma delirium then ioc and adn

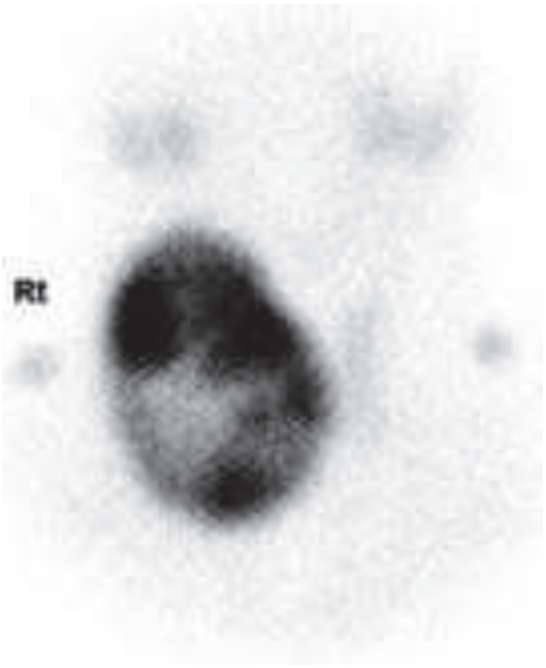
A 24-y hypot gestat medic adjust levoth





B

**s of Graves disease. (A)** Graves ophthalmopathy. **(B)** Pretibial myxedema. (Reproduce

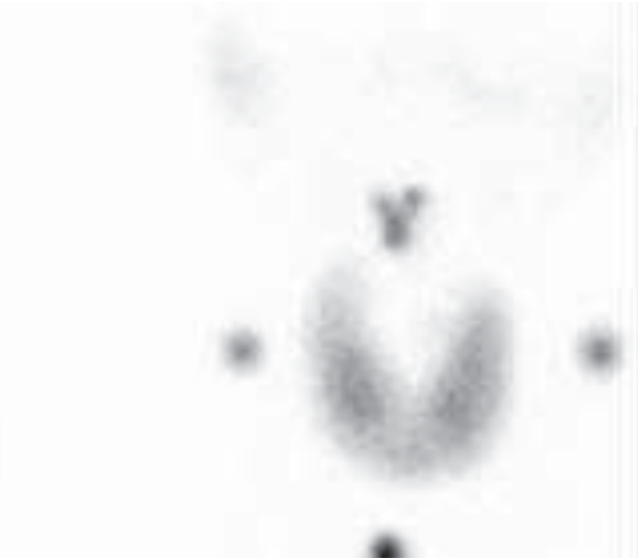



A

A

.
cy
TSH.





| | |
|---|---|
| | treating patients with Graves). **Contraindicated in pregnancy.** Will initially **worsen** ophthalmopathy. |
| | Hypothyroidism, hypoparathyroidism, damage to nearby structures (recurrent laryngeal nerve). |

SKI) is also effective. Untreated hyperthyroidism can also
**ne loss.**

levels of $T_3/T_4$. Most commonly due to **Hashimoto thy-**
ult from other causes (see Table 2.3-4).

**oiditis (autoimmune hypothyroidism):** Associated with
**ulin and antithyroid peroxidase (anti-TPO) antibodies**
nyroid destruction.
**tpartum, postviral, subacute):** Can have a hypothyroid
vs the hyperthyroid phase. Hypothyroidism can be perma-

**thyroidism:** Due to pituitary tumors or pituitary surgery.
**othyroidism:** Most common etiology is thyroid dysgene-
**ailure to thrive,** hypotonia, **umbilical hernias,** prolonged

**istance to thyroid hormone: Rare. Elevated** $T_3/T_4$, **nor-**
**TSH.**

eakness, fatigue, **cold intolerance, constipation,** weight
, hair loss, menstrual irregularities, myopathy, and **hoarse-**

ulomatous, radiation-induced, autoimmune, postpartum (eg, amiodarone) thyroiditis.

### History/Pe

- The **subacute form presents with a tender thyroid** symptoms.
- All other forms are associated with painless goiter.

### Diagnosis

Thyroid dysfunction (typically thyrotoxicosis followed b with ↓ uptake on RAI and scan during the hyperthyroid ph

### Treatment

- β-blockers for hyperthyroidism; levothyroxine for hypot
- Subacute thyroiditis is usually self-limited; for sever NSAIDs or with oral corticosteroids.

## THYROID NEOPLASMS

Thyroid nodules are very common and show an ↑ incide (~ 95%) are benign.

### History/Pe

- Usually asymptomatic on initial presentation; discover imaging for other purposes (eg, carotid ultrasound) or
- Hyperfunctioning nodules present with hyperthyroidis
- Large nodules adjacent to the trachea/esophagus prese toms (dysphagia, dyspnea, cough, choking sensation).
- An ↑ risk of malignancy is associated with a **history irradiation, "cold" nodules** (minimal uptake on RA age < 20 or > 70, firm and fixed solitary nodules, a ⊕

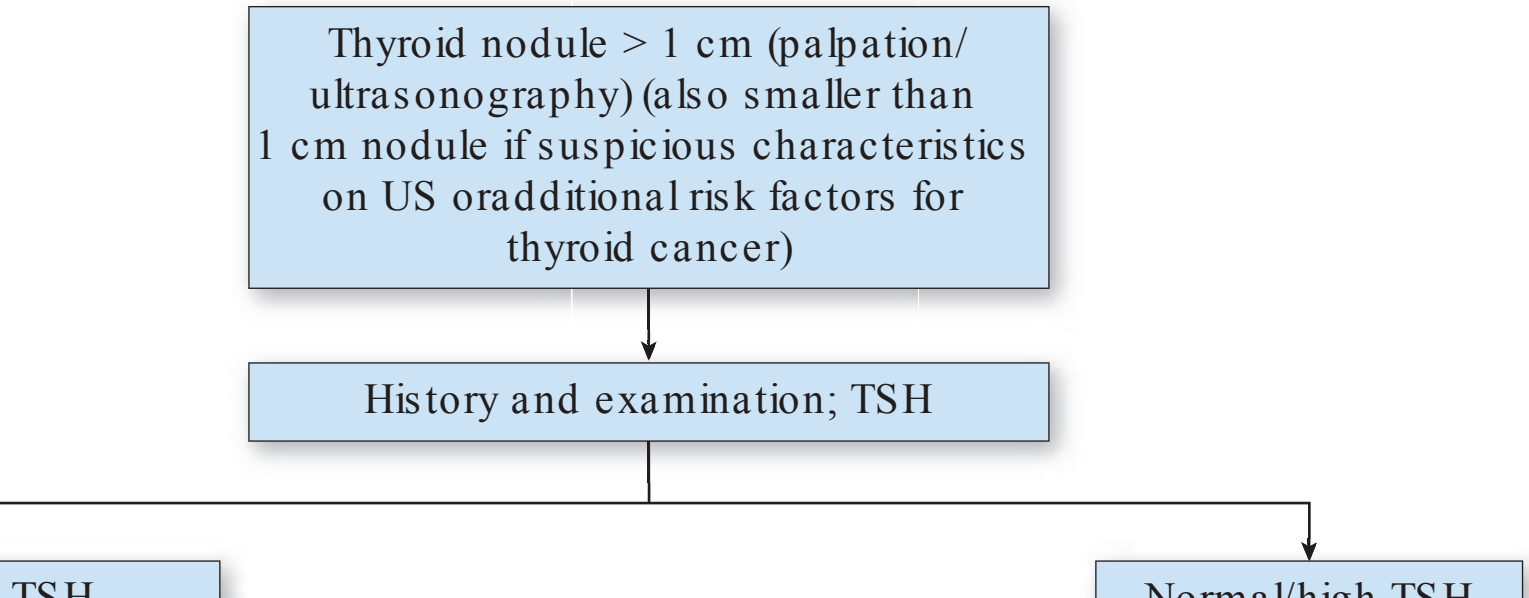
| | |
|---|---|
| nsible for 6–8% of thyroid cancers. Found in calcitonin-cing C cells; the prognosis is related to degree of lar invasion. | Eighty percent of patients su surgery. Consider MEN 2A or |
| nts for < 2% of thyroid cancers; rapidly enlarges and tasizes. | Ten percent of patients survi |

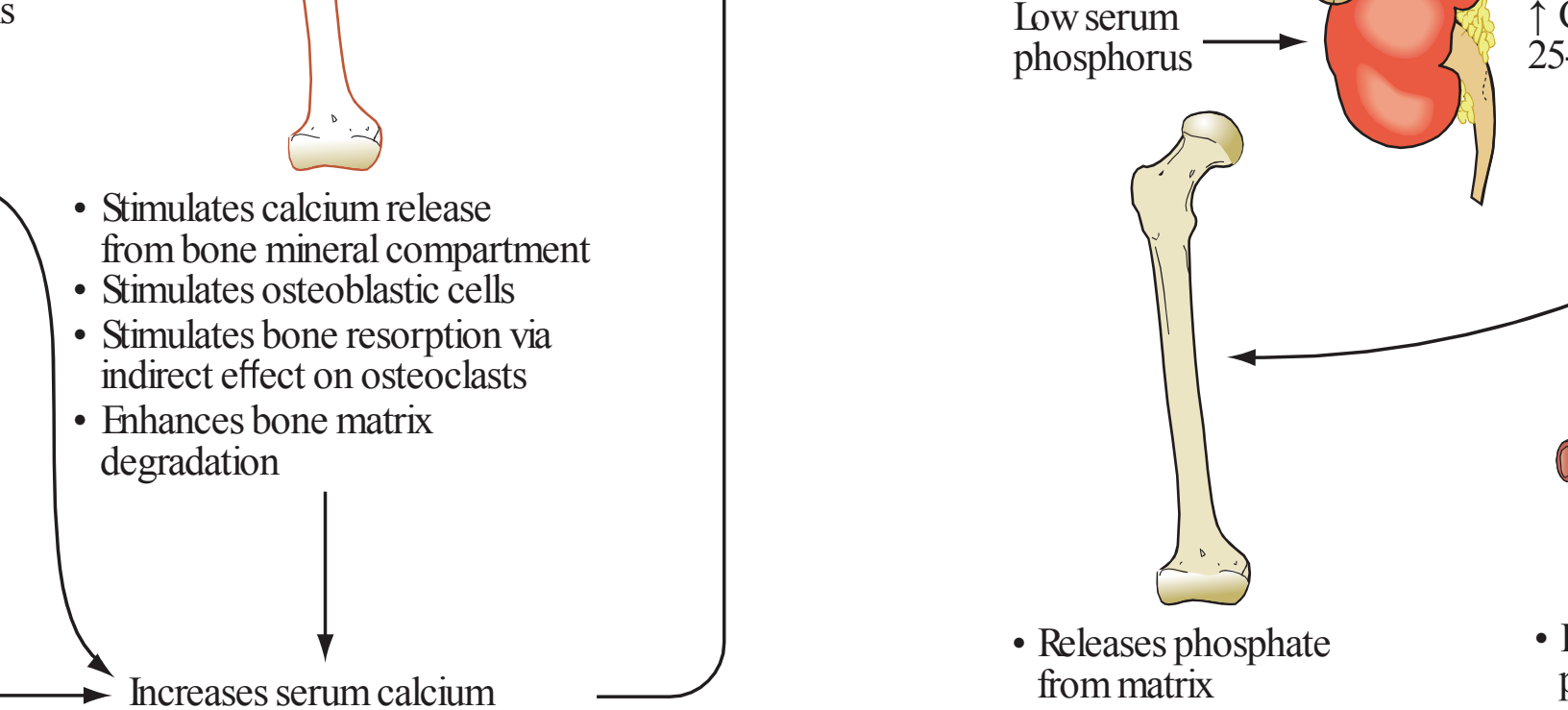
xed papillary and follicular pathologies.

**:** Surgical resection with hemi- or total thyroidectomy is nt; adjunctive radioiodine ablation following excision is ome follicular lesions.
**FNA:** Watchful waiting vs hemithyroidectomy (10–30% nancy). If resected, await final pathology to guide further

## l Mineral Disorders

calcium and phosphate regulation.


Thyroid nodule > 1 cm (palpation/ ultrasonography) (also smaller than 1 cm nodule if suspicious characteristics on US oradditional risk factors for thyroid cancer)
History and examination; TSH


• Stimulates calcium release from bone mineral compartment
• Stimulates osteoblastic cells
• Stimulates bone resorption via indirect effect on osteoclasts
• Enhances bone matrix degradation
Increases serum calcium
Low serum phosphorus
• Releases phosphate from matrix

Calcium homeostasis

Phosphate home

**calcium and phosphate regulation.** (Adapted with permission from Chandrosoma P et al. Concise

## OSTEOPOROSIS

A common metabolic bone disease characterized by low
often affects thin postmenopausal women (17%), especia
Asians, with risk doubling after age 65. Males are also at
but the diagnosis is often overlooked.

### History/Pe

- Commonly asymptomatic even in the presence of a ve
- Examination may reveal **hip fractures, vertebral co** (loss of height and progressive thoracic kyphosis), and/ tures (Colles fracture) following minimal trauma (see
- **Smoking, age,** excessive caffeine or alcohol intake, a depleting conditions in women (eg, amenorrhea,



**iographic findings in osteoporosis** Lateral thoracic spine radiograph
n anterior wedge deformity of a lower thoracic vertebral body with
s is a typical insufficiency fracture in osteoporotic patients. (Reproduced
t al. Harrison's Principles of Internal Medicine, 17th ed. New York, NY: McGraw-Hill; 2008.)

reatment with **calcium** and **vitamin D supplementation.**
on and weight-bearing exercises help maintain and even
e density.
**s** (eg, alendronate, risedronate, ibandronate, zoledronic
the treatment of osteoporosis, **not** osteopenia.
lude antiresorptive agents like selective estrogen recep-
eg, raloxifene), intranasal calcitonin, and denosumab (a
body to RANK-L), as well as anabolic agents like teripara-
f parathyroid hormone, PTH).

t devastating consequence of low BMD/osteoporosis, car-
**e in mortality** in the year following hip fracture.

n ↑ rate of bone turnover with both excessive resorption
ne, leading to a **"mosaic" lamellar bone pattern** on x-ray
be due to the effects of a latent viral infection in geneti-

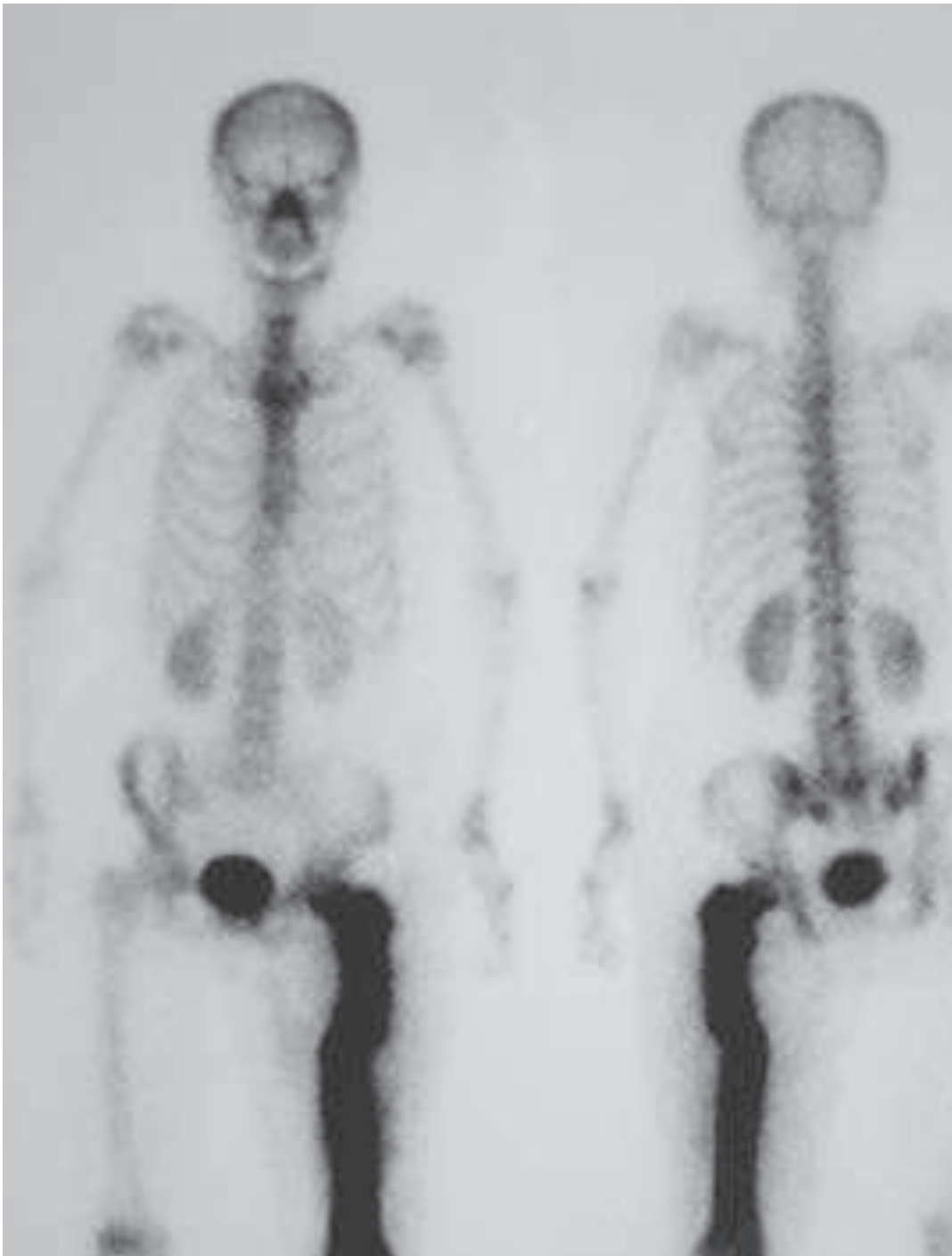


**hic fin** - ograph (arrow), ), and ad, clas- ed with nciples of -Graw-Hill;

- There is no cure for Paget disease. Treat with calci supplementation. In the setting of severe pain, involv able site (femoral neck), or fracture, **bisphosphonates** be used to slow osteoclastic bone resorption; NSAIDs can be given for pain management.

## Complications

Pathologic fractures, high-output cardiac failure (from AV sarcoma (up to 1%).



or vitamin D deficiency.

**roidism:** Seen in **dialysis patients** with long-standing 2° dism that leads to hyperplasia of the parathyroid glands. ore of the glands become autonomous, 3° hyperparathy-

**athyroidism:** PTH resistance. Elevated PTH levels but get organs. Hypocalcemia and hyperphosphatemia. Asso- ght hereditary osteodystrophy (may have shortened fourth tacarpal bones).

perparathyroidism are **asymptomatic** but may show signs **percalcemia** (see Renal/Genitourinary chapter).

° hyperparathyroidism reveal **hypercalcemia, hypophos-** ypercalciuria. Intact PTH is inappropriately ↑ relative to calcium (see Table 2.3-8).

al low BMD or frank osteoporosis in the distal radius or

i scan, in conjunction with thyroid ultrasonography, can olitary adenoma.

**my** if the patient is symptomatic or if certain criteria are n,↑ creatinine, ↓ BMD, age < 50). In the case of a soli- gland can be removed. In the setting of hyperplasia, 3.5 emoved.

**alues in Hyperparathyroidism**

Hyperca
"stones,
psychiat
fluids an
bisphos
± calcito

Etiologie
include
autoimm
infiltrati
diseases

Postparathyroidectomy, watch out for hungry bone syndro
longed **hypocalcemia** due to acute reversal of PTH and
calcium, phosphate, and magnesium).

# Pituitary and Hypothalamic Disorders

Figure 2.3-12 illustrates the hypothalamic-pituitary axis. T
low outline the manner in which the components of th
target organs in various pathologic states.

## HYPOPITUITARISM

Caused by mass lesions (tumors, cysts), surgery, radiation
infarction, often 2/2 postpartum hemorrhage), apoplexy
trative disorders (hemochromatosis), infections. Gonadc
hormone often affected first.

### History/Pe

See corresponding sections for presentation of pituitary ho
May present **suddenly** (apoplexy, Sheehan) or **graduall**
tive diseases).

### Diagnosis

Measure 8 AM cortisol, free $T_4$ (TSH not diagnostic), t
levels, insulin-like growth factor 1 (IGF-1).

### Treatment

Treat underlying disorder. See corresponding sections fo
cific hormone deficiencies.

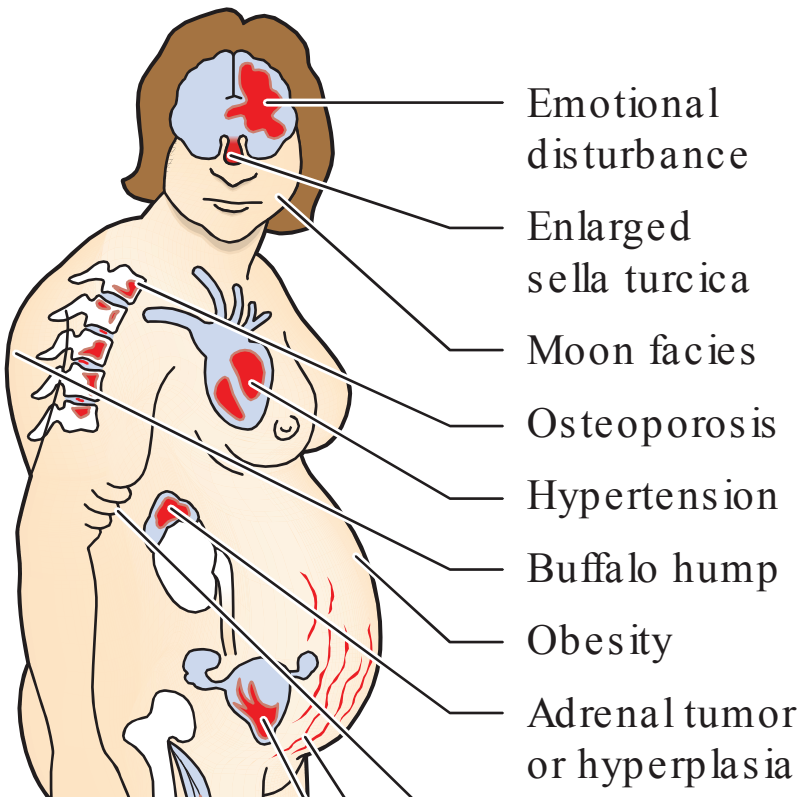


**r classic signs and symptoms of Cushing syndrome.**

stepwise progression of tests. See Figure 2.3-15 for the n. Table 2.3-9 outlines important lab findings that aid in

**on** of the source (pituitary, adrenal, neoplasm).
mone replacement therapy to correct deficiencies after 1° lesion.

rmone (GH) levels in adults, most commonly due to a H-secreting adenoma (see Figure 2.3-16). Children with on present with **gigantism.**

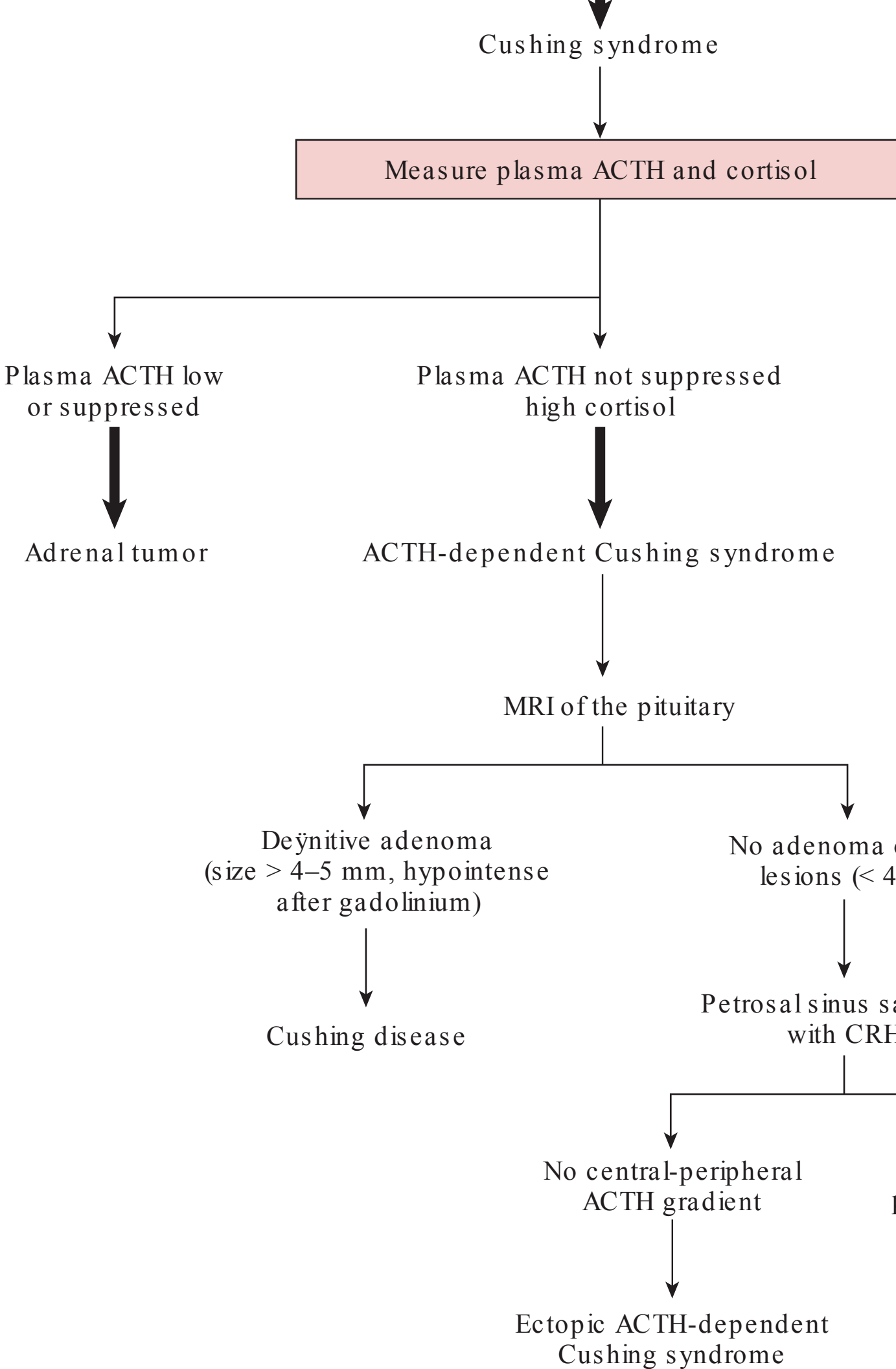



Poste

**FIGUR**
**pituitary**

**Cushing**
cortisol.
cortisol
pituitary

In Cushi
remains
(1 mg) d
suppres
dexame


Cushing syndrome
Measure plasma ACTH and cortisol
Plasma ACTH low
or suppressed
Plasma ACTH not suppressed
high cortisol
Adrenal tumor
ACTH-dependent Cushing syndrome
MRI of the pituitary
Deÿnitive adenoma
(size > 4–5 mm, hypointense
after gadolinium)
No adenoma
lesions (< 4
Cushing disease
Petrosal sinus sa
with CRH
No central-peripheral
ACTH gradient
Ectopic ACTH-dependent
Cushing syndrome

| | | | | |
|---|---|---|---|---|
| | ↑ | ↓ | ↑ | ↓ |
| | | N/A[a] | | N/A[a] |
| | ↑ | | ↑ | |
| | ↓ | | ↑ | |

ression test is not required once the diagnosis of ACTH-independent
de.

also lead to **glucose intolerance, diabetes, and cardiomy-**

**IGF-1 levels (↑ with acromegaly);** confirm the diagno-
glucose suppression test (GH levels will remain elevated
dministration). Baseline **GH is not a reliable test.**
nows a sellar lesion.

surgical resection or external beam radiation of the tumor.
nreotide (somatostatin analogues) can be used to suppress
egvisomant (a GH receptor antagonist) can be used to
eral actions of GH.

f death is from **cardiovascular complications** (CHF).

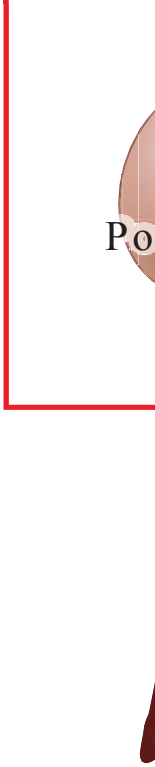



**FIGURE**
**pituitary**

Measure
acromeg

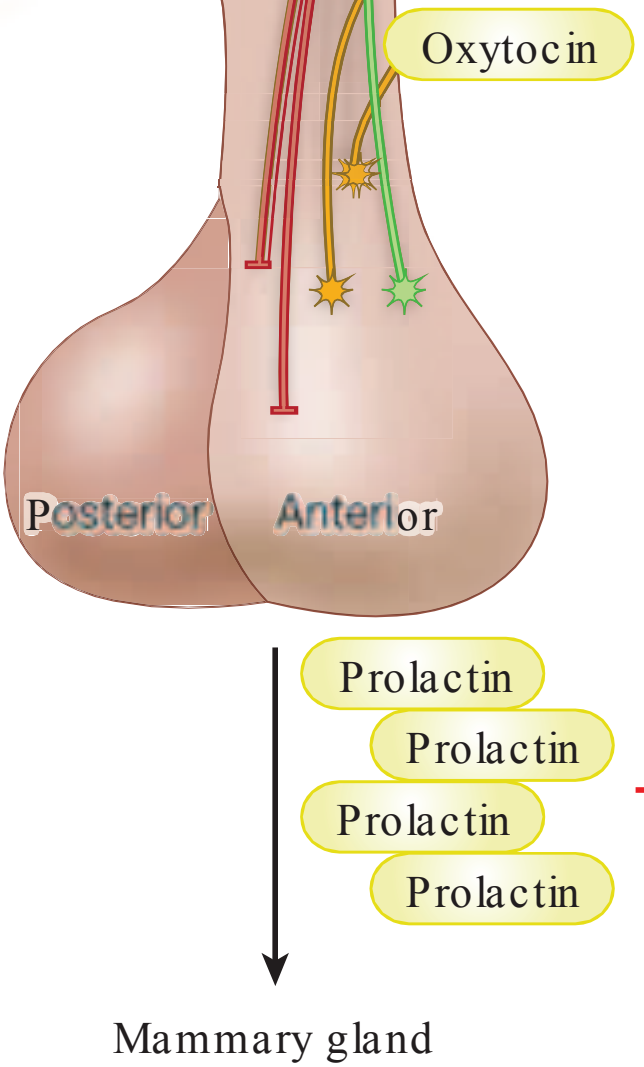



**FIGURE 2.3-17. The hypothalamic-pituitary axis: prolactin regu**

### Diagnosis

- The serum **prolactin level is typically > 200 ng/mL.**
- MRI shows a sellar lesion.

### Treatment

- **First line:** Dopamine agonists (eg, **cabergoline,** bromo
- **Surgery:** Indicated in adenomas refractory to medic with compressive effects (eg, visual loss).

## DIABETES INSIPIDUS (DI)

Inability to produce concentrated urine as a result of AD

c DI, patients excrete a high volume of **inappropriately**

**cetate (DDAVP, a synthetic analogue of ADH) replace-**

↓ urine output and ↑ **urine osmolarity** (by 50–100%).
**DI:** No effect is seen on urine output or urine osmolar-

pituitary or hypothalamic mass in central DI.

ying cause.
minister DDAVP intravenously, intranasally, or orally.
**I:** First-line treatment consists of salt restriction and water
diuretics can be used to promote mild volume depletion
↑ water reabsorption.

**FIGUR**
**pituitar**

of euvolemic hyponatremia that results from **persistent**
**endent of serum osmolality** (see Figure 2.3-19).

**S disease** (eg, head injury, tumor), **pulmonary disease**
, pneumonia), ectopic tumor production/paraneoplastic
cell lung carcinoma), and drugs (eg, antipsychotics, anti-
s).

y is > 50–100 mOsm/kg in the setting of serum hypo-
**ut a physiologic reason for ↑ ADH** (eg, CHF, cirrhosis,

**m level is often > 40 mEq/L.**

- **1°:** In the United States, most commonly due to au cortical destruction **(Addison disease).** Other causes enzyme deficiencies, adrenal hemorrhage (Waterhou drome from Neisseria meningitidis), and infections (H TB). TB is the most common cause of AI worldwide (c of the adrenal glands).
- **2°/3°:** Caused by ↓ ACTH production by the pituitary tion by the hypothalamus; **most often due to cessatio cocorticoid treatment (often > 20 g for > 3 weeks).**

### History/Pe

- Most symptoms are nonspecific.
- **Weakness, fatigue,** and **anorexia with weight loss** are
- **Hyperpigmentation** (due to ↑ ACTH secretion) is s ease.

### Diagnosis

**8 AM plasma cortisol levels** and **ACTH levels** can lead Table 2.3-10 for laboratory findings). A **synthetic ACTH tropin) test** is the test of choice if morning cortisol levels a

- An 8 AM plasma cortisol level < 3 µg/dL in the absenc cocorticoid administration is diagnostic of AI.
- Failure of cortisol to rise > 20 µg/dL following ACTH firms the diagnosis.

### Treatment

- **1°:** Glucocorticoid and **mineralocorticoid** replacemen
- **2°/3°:** Only glucocorticoid replacement is necessary **production is not ACTH dependent).**
- In adrenal crisis, provide IV steroids; correct electrol

50% dextrose to correct hypoglycemia; and initiate aggres-
scitation.
periods of stress (eg, major surgery, trauma, infection).
ronic steroid therapy, taper slowly to prevent 2°/3° AI.

## OMA

fin tissue that secretes catecholamines and is found either
ulla or in extra-adrenal sites. Most commonly associated
**2B.**

**roxysmal** tachycardia, palpitations, chest pain, diaphore-
, headache, tremor, and anxiety.
obtain a family history in order to rule out genetic causes
cytoma (eg, MEN 2A/2B, von Hippel–Lindau disease,
is).

e both sensitive for pheochromocytomas (see Figure 2.3-
MIBG scan can localize extra-adrenal lesions and meta-

d plasma-free metanephrines (metanephrine and normeta-
hour urine metanephrines and catecholamines.

n.
use α-adrenergic blockade first (phenoxybenzamine)
rtension, followed by β-blockade to control tachycardia.
**ockade first,** as unopposed α-adrenergic stimulation can
pertension.

10% Extr
10% Bila
10% Mal
10% Occ
10% Fam

The 5 P

**P**ressure
**P**ain (hea
**P**erspirat
**P**alpitati
**P**allor

Q

A 23-y
of sch
compl
cramp
past se
any ot
he had
while
reveal
depriv
What

A

B




**FIGURE 2.3-21. Pheochromocytoma. (A)** MRI showing left supra
chromocytoma postsurgical resection. (Reproduced with permission from Roghi A
in pheochromocytoma, J Cardiovasc Magn Reson 2011 Jan 11;13:4.)

## Diagnosis

- Labs show **hypokalemia,** metabolic **alkalosis,** hypom
  dosteronism, and an ↑ **aldosterone-to-plasma renin a**
  > 30).
- CT or MRI may reveal an adrenal mass. Adrenal veno
  needed to localize the adenoma or to confirm bilater
  sia.

## Treatment

- Surgical resection for adrenal tumors (after correctio
  sium).
- Treat bilateral hyperplasia with an aldosterone recepto
  **none** preferred over spironolactone as has fewer side ef

| | | | | | |
|---|---|---|---|---|---|
| | ↓ | ↑ | ↓ | ↑ | ↑ renin activity<br>↑ 17-hydroxy-progesterone |
| ↓ aldosterone<br>↑ 11-deoxycorti-costerone (results in ↑ BP) | ↓ | ↑ | ↑ | ↓ | ↓ renin activity |

nzyme deficiencies a e characterized by an enlargement of both adrenal glands due to

ssion from Le T, Bhushan V. First Aid for the USMLE Step 1 2015. New York, NY: McGraw-Hill

rmalities include hyponatremia, hyperkalemia, and meta-
n severe cases, mineralocorticoid deficiency may lead to
alt wasting.

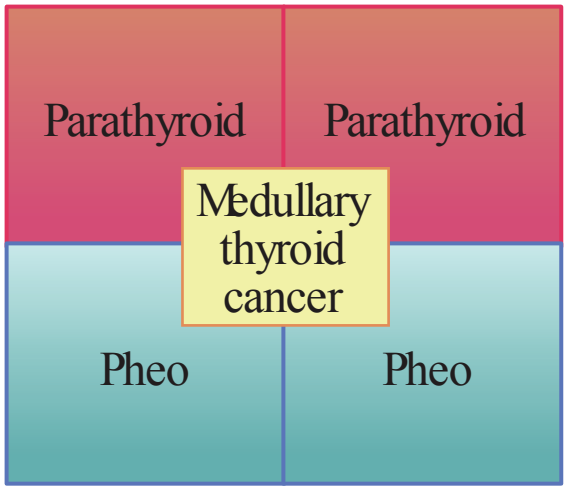
**erum 17-hydroxyprogesterone level** is diagnostic of
eficiency.

**diate fluid resuscitation and salt repletion.** Administer
TH and adrenal androgens. Fludrocortisone is appropriate
roxylase deficiency.

**l correction of ambiguous genitalia.**

necology chapter for information on the diagnosis and
-onset CAH.

MEN 2A = **2 P's**: **P**arathyroids and **P**heochromocytoma; remember by drawing square.




MEN 2B = **1 P**: **P**heochromocytoma; remem by drawing a triangle.

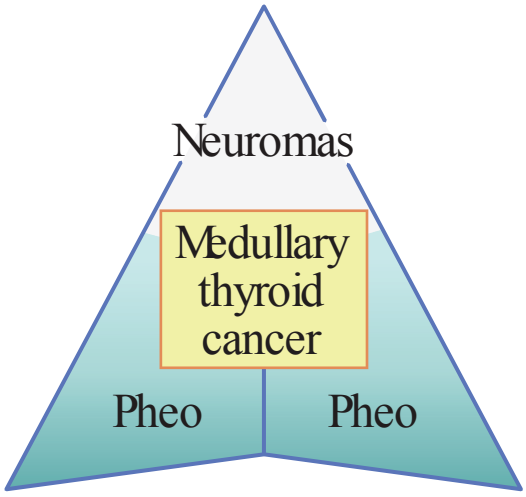



**FIGURE 2.3-22. Multiple endocrine neoplasias (MEN).** (Reproduced
Bhushan V. First Aid for the USMLE Step 1 2015: A Student-to-Student Guide. New York, NY: McGra

- **MEN type 2A (Sipple syndrome):** Medullary carcin pheochromocytoma or adrenal hyperplasia, parathyroi Due to mutations in the **RET proto-oncogene.**
- **MEN type 2B:** Medullary carcinoma of the thyroid, p oral and intestinal ganglioneuromatosis (mucosal ne habitus. Due to mutations in the **RET proto-oncogene**

Frequency 112
stic Studies 112
Y 112
REd Ic TIVE VALUES 113
113
113
114
es 114
s 115
115
116
TRIALS 116
Evaluating Clinical Studies
BIAS
c HANc E
PREVENTIo N
VAc c INATIo N
Behavioral Counseling
Screening Recommendations
Causes of Death
Reportable Diseases

$$\text{Incidence} = \frac{\text{number of } \textbf{new} \text{ cases in the population over}}{\text{total population at risk during the specified}}$$

- Prevalence depends on incidence and duration:

  Prevalence (P) = incidence (I) × average duration

- For incidence, remember to subtract any preexisting from the total population at risk, as these individuals ar

## Assessment of Diagnostic Studies

### SENSITIVITY AND SPECIFICITY

Physicians often use tests to narrow and confirm possible **sitivity** and **specificity** of these tests allow physicians to d false positives and negatives occur (see Figure 2.4-1).

- **Sensitivity:** The probability that a patient with a disea result.
  - A sensitive test will **rarely miss people** with the dis good at **ruling out those who do not have the dise**
  - Desirable early in a **diagnostic workup or scree** necessary to reduce a broad differential diagnosis.
  - **Example:** An initial ELISA test for HIV infection.

    False-⊖ ratio = 1 – sensitivity

- **Specificity:** The probability that a patient without a d test result.
  - A specific test will rarely determine that someone h in fact they do not and is therefore good at **ruling the disease.**
  - Desirable when **confirming a likely diagnosis.**

ability that a patient with a ⊖ test result truly **does not . The lower the disease prevalence, the higher the NPV at disease.**

ent to which a given test result is likely in diseased people e without disease:

v much the odds (or probability) of disease are ↑ if the test

v much the odds (or probability) of disease are ↓ if the test

$$\text{Posttest odds} = \text{pretest odds} \times \text{LR}$$

## s of Effec

ays to express and compare risk. These include the follow-

ne incidence of disease.

**x (or risk difference):** The difference in risk between the xposed groups.

ncidence of disease in exposed − incidence in unexposed.

**l to treat:** Number of individuals that need to be treated enefit.

er need to treat (NNT) = 1 / attributable risk

**studies.** The OR tells how much more likely it is that ease has been exposed to a risk factor than someone **The lower the disease incidence, the more closely i** In case-control studies, the OR also describes how mar an exposed individual is to have disease compared to vidual (see Figure 2.4-3).

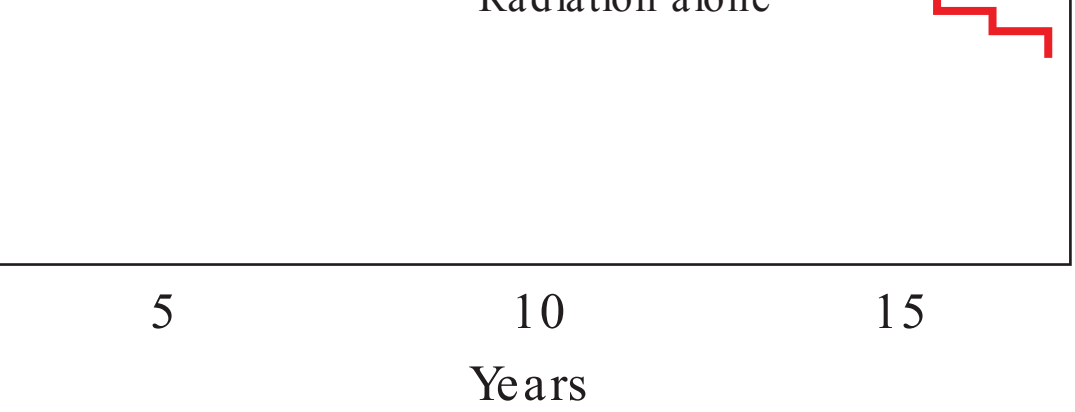
$$\text{Odds ratio} = \frac{\text{odds that a diseased person is ex}}{\text{odds that a nondiseased person is}}$$

## Survival Curves

Once a diagnosis has been established, it is important to the associated prognosis. **Survival analysis is used to su age time from one event** (eg, presentation, diagnosis, or s **any outcome that can occur only once during follow-up** rence of cancer). The usual method is with a **Kaplan-Mei** 2.4-4) describing the survival in a cohort of patients, wit survival decreasing over time as patients die or drop out fr

## Types of Clinical Studies

Studies are typically used to evaluate diagnosis, treatmen a disease. Although **the gold standard for such evaluatio double-blinded controlled trial,** other types of studies m (eg, an observational study, in which the exposure in que tic intervention). In descending order of quality, publishe treatment options include randomized controlled trials (R studies, and case series/case reports. Meta-analyses are o atically synthesize information across studies to help summ




**mple of a Kaplan-Meier curve.**

## STUDIES

udy is an observational study that assesses risk factors and shot in time. These studies aren't able to prove temporal se they measure correlation, not causation.

of cross-sectional studies are as follows:
an efficient means of examining a population, allowing assessment of people with the disorder and those without

used as a basis for diagnostic testing.
**ages** include the following:
determine causal relationships, because information is at a **single point in time.**
cidence of disease cannot be directly measured.

A cross-
underta
called a

a group of people is assembled, **none of whom has the st** (ie, the disease), but **all of whom could potentially tcome.** For each possible risk factor, the members of the as either exposed or unexposed. All the cohort members ver time, and the **incidence of outcome events is com-**

A loca
before
elevat
studer
schoo
event

## CASE-CONTROL STUDIES

In **case-control studies,** a series of **cases** are identified **trols** are sampled from the underlying population to esti of exposure in the population at risk of the outcome. researcher **compares the frequency of exposure to a** between the case and control groups. **Example:** A stud with heart disease (cases) and without heart disease (con exposures to red meat between both groups.

- The validity of a case-control study depends on appr cases and controls, the manner in which exposure is manner in which extraneous variables (confounders) a
- Cases and controls should be comparable in terms of c sure (ie, they should be members of the same base equal opportunity of risk factor exposure).
- **"Matching"** in case-control studies occurs when the controls that match cases on a particular character if matching on gender, female cases would be matc trols and male cases would be matched to male contr matching is to ↓ confounding.
- **Advantages** of case-control studies are as follows:
  - They use smaller groups than cohorts, thereby redu
  - They can be used to study rare diseases and can e tiple risk factors.
- **Disadvantages** include the following:
  - Studies cannot calculate disease prevalence or in estimate the relative risk, because the numbers o without a disease are determined artificially by th than by nature; **however, an OR can be used to est RR, if the prevalence is low.**
  - Retrospective data can be inaccurate as a result of r biases.

**uses results to systematically differ from the truth.** Com-
clude the following:

**arison of Study Designs**

| rct | Cohort | Cross-Sectional |
|---|---|---|
| ts causality through random gnment of exposure. | Follows groups of patients over a specified pe iod of time to capture the association of risk factors to the development of disease. | Determines prevalence in a snapshot of time. |
| measures that are reported gest causal relationships. | RR, OR, incidence, prevalence. | Prevalence (not incidence). |
| jects are randomly assigned e in exposed (treatment A) nexposed (treatment B) ups. | Subjects are not assigned to groups.<br>Determines if subjects are in exposed or unexposed groups and follows them until they develop the disease (or do not). | Determines disease prevalence at 1 point in time; cannot determine the directionality of association between exposure and outcome |
| determine causality; imizes bias and confounding. | Temporality can be determined; incidence can be determined. | Less time consuming and costly. |

experience a higher incidence of lung cancer than th
eral population. However, if smokers are more likely t
and are also more likely to develop lung cancer than
ing a fisherman will not in itself lead to lung cancer. R
ing to which those fishermen are exposed that causes t

- **Effect modification,** on the other hand, occurs able disproportionately affects two groups. Effect a meaningful difference, whereas confounding A new chemotherapeutic agent is shown to impr Hodgkin lymphoma, but only in patients who are u therapy, whereas those not receiving radiation show

- **Recall bias:** Results from a difference between two spective recall of past factors or outcomes. **Example:** A may be more motivated than would a healthy individu sodes of chemical exposure.
- **Lead-time bias:** Results from earlier detection of disea ance of prolonged survival when in fact the natural c **Example:** A new and widely used screening test that d earlier may yield the impression that patients are living ease.
- **Length bias:** Occurs when screening tests detect a dis ber of slowly progressive diseases but miss rapidly prog to overestimation of the benefit of the screen. **Exampl** for patients with cancer is celebrated following the i new screening program. However, this test disproportio growing tumors, which generally tend to be less aggres

## CHANCE

Even with bias reduction, unsystematic random error is u of chance variation in studied data. Types of errors are as f

- **Type I (α) error:**

g a 95% CI, there is a **95% chance that the interval con-**
**e value.**
**le sizes produce more power and narrower CIs.** If the
he null value (RR or OR of 1.0 or 0%), the results are not
gnificant.
RCT studying aspirin to prevent myocardial infarction
ve risk of 0.9 with a 95% CI of 0.85 to 0.95 in a sample of
, whereas in a sample of 30 patients the 95% CI is 0.1 to
example shows a significant difference, whereas the sec-

evels of prevention:
**n:** Includes preventive measures to ↓ the incidence of dis-

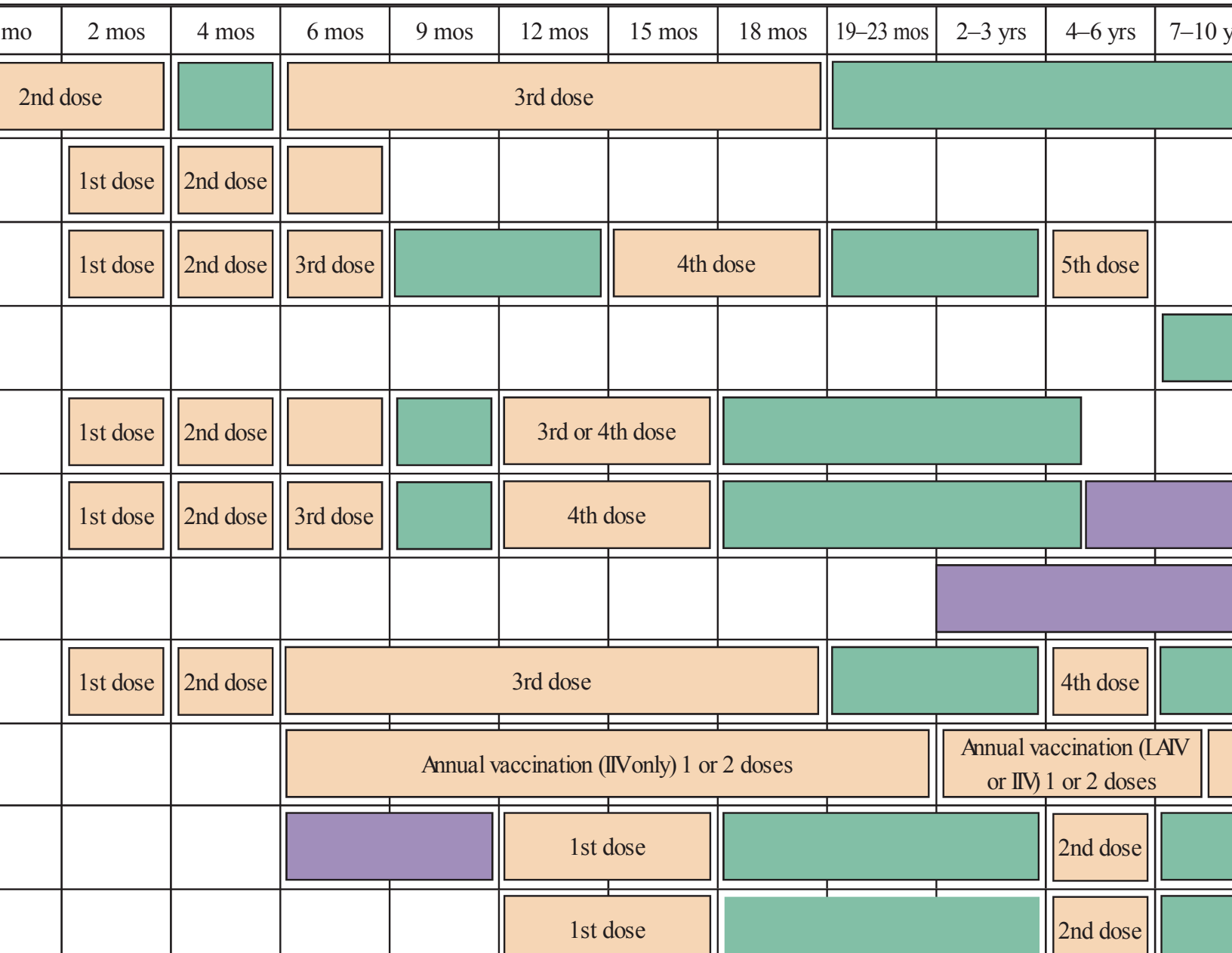
**n:** Focuses on identifying the disease early, when it is
c or mild, and implementing measures that can halt or
progression. Includes screening tests that are designed to
inical disease.
**n:** Includes measures that ↓ morbidity or mortality result-
presence of disease.
be accomplished by a **combination of immunization,**
**n, behavioral counseling, and screening. A good screen-**
ollowing characteristics:
**ensitivity and specificity** (usually more important to have
ty to **rule out** those who don't have the disease).
**PPV.**
ive, easy to administer, and safe.
ter screening is more effective than subsequent treatment
ning.

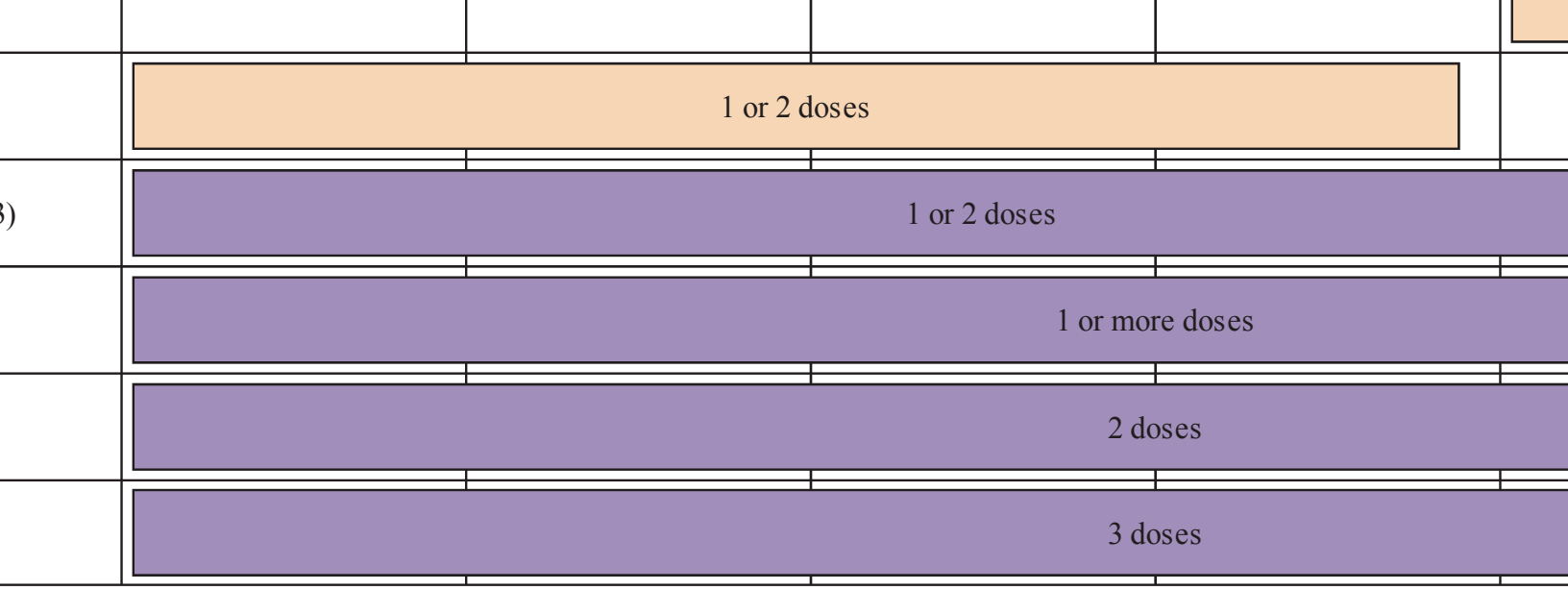
- **1° p**
  diet
  redu
  canc
- **2° p**
  a ma
  canc
- **3° p**
  und
  tam

| | |
|---|---|
| Subunit | HBV, pertussis, Streptococcus pneumoniae, |
| Conjugate | Hib, S pneumoniae. |

## Behavioral Counseling

In offering counsel, physicians should tailor their educat
to the individual patient as well as to his or her **stage of**
2.4-3).






gory who meet the age requirements and who ination or have no evidence of previous infec- mended regardless of prior episode of zoster

Recommended if some other risk factor is present (eg, on the basis of medical, occupational, lifestyle, or other indication)

**ommended vaccinations for adults.** (Reproduced with permission from the Centers for Disease Co es/downloads/adult/adult-combined-schedule.pdf. Data from 2015.)

## s of Change in Behavioral Counseling

| | Characterization |
|---|---|
| Denial or ignorance of the problem. | A heroin addict has not |
| Ambivalence or confli ted emotions; assessing benefits and barriers to change. | The heroin addict consid |
| Experimenting with small changes; collecting information about change. | The heroin addict visits h quitting. |
| Taking direct action toward achieving a goal. | The addict enters a rehal addiction. |
| Maintaining a new behavior; avoiding temptation. | The addict continues to |

| ular | breaSt/reproductiVe | o |
|---|---|---|
| t once every<br><br>g starting at<br>s at ↑ risk of | Pap test every 3 years starting at age 21; co-testing (Pap + HPV) may be done every 5 years starting at age 30.<br>Chlamydia test yearly until age 24 if sexually active. Women ≥ 25 should be tested only if there is an ↑ risk.<br>HIV test at least once to ascertain status.<br>Test for gonorrhea and syphilis if at increased risk. | **Diabetes:** Blood gluc<br>starting if BP > 135/80<br>medication for hypert |
| t once every<br><br>g for<br>sed risk of | Pap test every 3 years or co-testing every 5 years.<br>Pelvic examination yearly; chlamydia test if the patient has new or multiple partners.<br>HIV test at least once to ascertain status.<br>Test for gonorrhea and syphilis if at increased risk. | **Diabetes:** Blood gluc<br>starting if BP > 135/80<br>medication for hypert |
| t once every<br><br>g for<br>sed risk of | Mammogram once every 1–2 years (can start at age 40, if patient chooses).<br>Pap test every 3 years.<br>Chlamydia test if the patient has new or multiple partners.<br>HIV test at least once to ascertain status.<br>Test for gonorrhea and syphilis if at increased risk. | **Diabetes:** Blood gluc<br>starting if BP > 135/<br>medication for hyp<br>**Bone:** Discuss BMD te<br>nurse.<br>**Colorectal:** FOBT yea<br>sigmoidoscopy eve<br>colonoscopy every |
| t once every | Mammogram once every 1–2 years until the | **Diabetes:** Blood gluc |

| | | |
|---|---|---|
| g at least once every 2 years.<br>screening for all men over<br>f 35. | Discuss DRE and PSA with physician or nurse.<br>HIV test at least once to ascertain status.<br>Test for syphilis if at increased risk. | **Diabetes**<br>screening<br>or taking |
| g at least once every 2 years.<br>screening for all men over<br>f 35. | Discuss DRE and PSA with physician or nurse.<br>HIV test at least once to ascertain status.<br>Test for syphilis if at increased risk. | **Diabetes**<br>screen<br>mm Hg<br>hypert<br>**Colorect**<br>sigmoi<br>colonc |
| g at least once every 2 years.<br>screening for all men over<br>f 35. | Discuss DRE and PSA with physician or nurse.<br>Discuss HIV test with physician.<br>Test for syphilis if at increased risk. | **Diabetes**<br>screen<br>mm Hg<br>hypert<br>**Colorect**<br>every 5<br>10 yea<br>with p<br>**Abdomi**<br>screen<br>smoke |

n from the US Department of Health and Human Services, Washington, DC, http://www
ations/screening-tests-for-men.pdf.

| | | |
|---|---|---|
| 5 | Stroke (4.2%) | Alzheimer diseas |
| 6 | Diabetes (3.1%) | Unintentional inj |
| 7 | Suicide (2.5%) | Diabetes (2.8%) |
| 8 | Alzheimer disease (2.0%) | Influenza and pn |
| 9 | Influenza and pneumonia (2.0% | Kidney disease (1 |
| 10 | Kidney disease (1.8%) | Septicemia (1.5% |

Adapted with permission from the Centers for Disease Control and Preven
www.cdc.gov/women/lcod/2011/WomenAll_2011.pdf. Data from 2011.

## Reportable Diseases

By law, disease reporting is mandated at the state level, an
that must be reported to public health authorities varies s
CDC has a list of nationally notifiable diseases that states vol
CDC. These diseases include but are not limited to those

**TABLE 2.4-7. Common Reportable Diseases**

| Disease Category | Examples |
|---|---|
| STDs | HIV, AIDS, syphilis, gonorrhea, chlamydia, cha |
| Tick-borne disease | Lyme disease, ehrlichiosis, Rocky Mountain s |
| Potential bioweapons | Anthrax, smallpox, plague. |

126 Disclosure
126 Full Disclosure
126 Medical Errors
Clinical Research
sion-Making Capacity 127 Confidentiality
128 Conflict of Interest
IVES 128
128 Malpractice
N-Assisted Suicide 128
128
128

saving transfusion to a Jehovah's Witness **(benefice**
patient's **autonomy** if he/she should refuse.

- **Nonmaleficence:** "Do no harm." All medical interve
fits and risks, and physicians should generally only rec
where the likely benefits outweigh the known risks.
  - **Example:** A surgeon declines to perform a proc
thinks the patient will die intraoperatively.
- **Justice:** Health care is a scarce resource. Fairness and
tion and delivery of health care are ongoing challeng
and in the clinical arena.

## Informed Consent

- Defined as willing and voluntary acceptance of a med
a patient after adequate discussion with a physician
the intervention along with its **indications, risks, ben
alternatives** (including no treatment).
- **Patients may change their minds at any time.**
- Informed consent is required for significant procedures
  - Emergency treatment is required.
    - **Examples:** An unconscious patient presents w
after a motor vehicle collision, or a patient with
cated DNR/DNI (do not resuscitate/do not int
goes cardiac arrest.
  - Patient lacks decision-making capacity (consent
must be obtained from a surrogate decision mak
assent should be obtained from the patient lacking
    - **Examples:** Patients may present with dementi
chiatric disturbances. Minors generally require
makers until they demonstrate adequate decisi
or are of legal age.

those decisions do not pose a serious threat to the child's
efusing immunizations is not considered a serious threat).
ision is not in the best interest of the child, a physician
tment against parental wishes. In emergent situations, if
tment jeopardizes the child's safety, treatment can be ini-
is of legal precedent.
physician provides blood transfusion to save the life of a
ild seriously injured in a motor vehicle collision despite
ests to withhold such a measure.

## nce and Decision-Making Capacity

person's **global and legal capacity to make decisions**
ountable in a court of law. **Competence is assessed by the**
inct from the term **decision-making capacity.**
**g capacity:** The ability of a patient to understand relevant
reciate the severity of the medical situation and its conse-
unicate a choice, and deliberate rationally about his/her
to the decision being made. This can be **assessed by the**

g capacity is best understood as varying with the complex-
n involved.
e level of capacity needed for a decision about liver trans-
different from that needed to choose between two types of
ion for fracture-related pain.
ients, as assessed by the courts, or temporarily incapaci-
ay still be able to provide assent for treatment or refuse
ever, the need to treat supersedes the refusal of an inca-
in emergency situations.
extremely hypertensive patient with altered mental sta-
es treatment must receive antihypertensive therapy, as this

Q

A47-y
with p
diagn
discus
any in
would
and ch
is the
manag

Q

A5-ye
needs
ventric
no sat
to relie
conse
arguin

- **Durable power of attorney for health care:** Legally de
  health care decision maker if a patient lacks decisio
  **More flexible than a living will.** Surrogates should ma
  tent with the person's stated wishes.
- If no living will or durable power of attorney for hea
  sions should be made by close family members (spo
  parents, and adult siblings) or friends, in that order.

## WITHDRAWAL OF CARE

- Patients and their decision makers have the right to
  life-sustaining treatment. Nevertheless, physicians sh
  stand patients and their reasons for refusing beneficial
- No ethical distinction is made between **withholdi**
  **withdrawing** a treatment, because a patient may choos
  vention either **before** or **after** it is initiated. This can
  fluids, nutrition, and medications such as antibiotics.
- If the intent is to relieve suffering and medication
  titrated for that purpose, it is considered ethical to pro
  ment to relieve pain and suffering even if it may hast
  **(principle of double effect).**
  - **Example:** A physician may prescribe an ↑ dose of
    to a patient who is expected to die within a day,
    suppress respiration and hasten death.

## EUTHANASIA AND PHYSICIAN-ASSISTED SUICIDE

- **Euthanasia** is the administration of a lethal agent with t
  - It is opposed by the AMA Code of Medical Ethics
    **states.**
  - Patients who request euthanasia should be evalu

right to know about their medical status, prognosis, and
s (full disclosure). They have the legal right to obtain cop-
cal records.
y cannot require that a physician withhold information
without the knowledge and consent of the patient.
withhold information only if the patient requests not to
e rare and controversial case in which a physician deter-
osure would cause severe and immediate harm to the
**utic privilege).**

**bligated** to inform patients of mistakes made in their med-

a specific error or series of errors is not known, the phy-
mmunicate this with the family promptly and maintain
patient as investigations reveal more facts.

H

bligated to inform patients considering involvement in a
protocol about the purpose of the research study and the
gn as it will affect the patient's treatment. This includes
, benefits, and alternatives to the research protocol.
nsent form approved by the overseeing research institu-
ard must be completed for participation in any clinical
l.

## tialit

losed by a patient to his/her physician and information

**Signs o**

- Hist
  inju
- Dela
- Sub
- Reti
- Spir
  fract
- Inju
  hea
- Unu

Overric

**WAIT a**
**dange**

**W**ounds
**A**utomol
**I**nfectiou
**T**arasof-
**S**uicide
**E**lder mis
**C**hild abu

months.

## Conflict of Interes

- Occurs when physicians find themselves having a pe
  given situation that influences their professional obliga
  - **Example:** A physician may own stock in a pharm
    (financial interest) that produces a drug he is presc
    (patient care interest).
- Physicians should disclose existing conflicts of interes
  (eg, patients, institutions, audiences of journal article
  ings).

## Malpractice

The essential elements of a civil suit under negligence inc

- The physician has a **Duty** to the patient.
- **Dereliction** of duty occurs.
- There is **Damage** to the patient.
- Dereliction is the **Direct** cause of damage.

Unlike a criminal suit, in which the burden of proof
able doubt," the burden of proof in a malpractice suit is "
the evidence."

A 132

132

133

GEAL SPASM 133

134

134

134

UX d ISEASE 135

136

ach and Duodenum 137

137

138

138

Ro ME 139

Bowel 140

140

ESTIo N 142

142

142

ME 143

N 143

144

145

145

Bowel 146

146

N 146

co Lo REc TAL c ANc ER

ISc HEMIc c o LITIS

Gastrointestinal Bleeding

Inflammatory Bowel Disease

Hernias

Biliary Disease

c Ho LELITHIASIS ANd BILIARY c o LIc

Ac UTE c Ho LEc YSTITIS

c Ho LEd o c Ho LITHIASIS

Ac UTE c Ho LANGITIS

GALLSTo NE ILEUS

Liver Disease

ABNo RMAL LIVER FUNc TIo N TESTS

HEPATITIS

c IRRHo SIS

PRIMARY Sc LERo SING c Ho LANGITIS

PRIMARY BILIARY c IRRHo SIS

No N-ALc o Ho LIc FATTY LIVER d ISEASE

HEPATo c ELLULAR c ARc INo MA

HEMo c HRo MATo SIS

WILSo N d ISEASE (HEPATo LENTIc ULAR d

Pancreatic Disease

INSULINo MA

VIPo MA

PANc REATITIS

PANc REATIc c ANc ER

- **Oropharyngeal dysphagia:**
  - Problem with **initiation of swallowing,** may involv into the lungs, leading to coughing, choking, or dro
  - **Etiology:** Neurologic or muscular, including stroke myasthenia gravis, prolonged intubation, and Zenk
  - Usually more of a problem with **liquids than with**
- **Esophageal dysphagia:**
  - Problem is with an **obstruction** (includes strictu webs, carcinoma) or **motility disorder** (include derma, esophageal spasm).
  - Obstructions usually more of a problem with **solid** while motility disorders cause both solid and liquid
  - Examine for masses (eg, goiter, tumor) and anatom

with
sitis

## Diagnosis

- **Oropharyngeal dysphagia:** Initially, modified barium roscopic swallowing exam). Occasionally esophago (EGD).

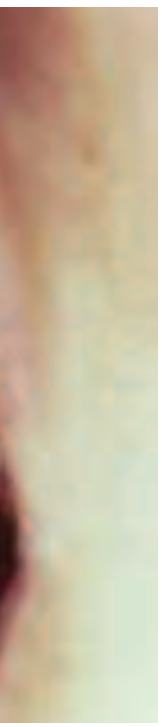


**TABLE 2.6-1. Causes of Infectious Esophagitis**

| Etiologic Agent | Exam Findings | Upper Endoscopy |
|---|---|---|
| Candida albicans | ± oral thrush (see Figure 2.6-1). | Yellow-white plaques adherent to the mucosa. |
| Herpes simplex virus | Oral ulcers. | Small, deep ulcerations; multinucleated giant cells with |

Treat the underlying disorder. Minimize food aspiration.

## HAGITIS

**ompromised** patients. Table 2.6-1 outlines the etiology, nent of infectious esophagitis.

Candida
defning

## ESOPHAGEAL SPASM

which normal peristalsis is periodically interrupted by **peristaltic** contractions (see Figure 2.6-2A).

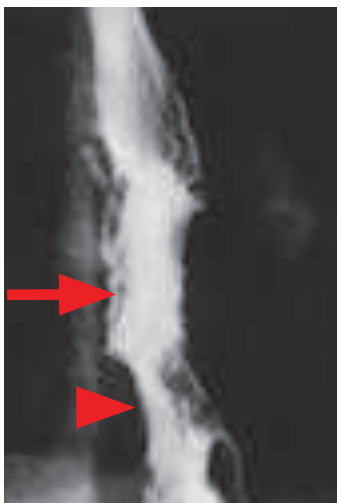
urn, chest pain, dysphagia, and odynophagia. **Often pre-on of hot or cold liquids; relieved by nitroglycerin.**

The mus
of the es
that of t
muscle.

structural abnormalities (see Dysphagia section above).
**: Corkscrew-shaped esophagus,** for very specific suspi-

**nometry (definitive test):** High-amplitude, simultaneous reater than 20% of swallows.







tal two-thirds of the esophagus.
- **Etiology:** Degeneration of the inhibitory neurons in t bach) plexus.

### History/Pe

Progressive dysphagia (solids and liquids), chest pain, re gested food, weight loss, and nocturnal cough.

### Diagnosis

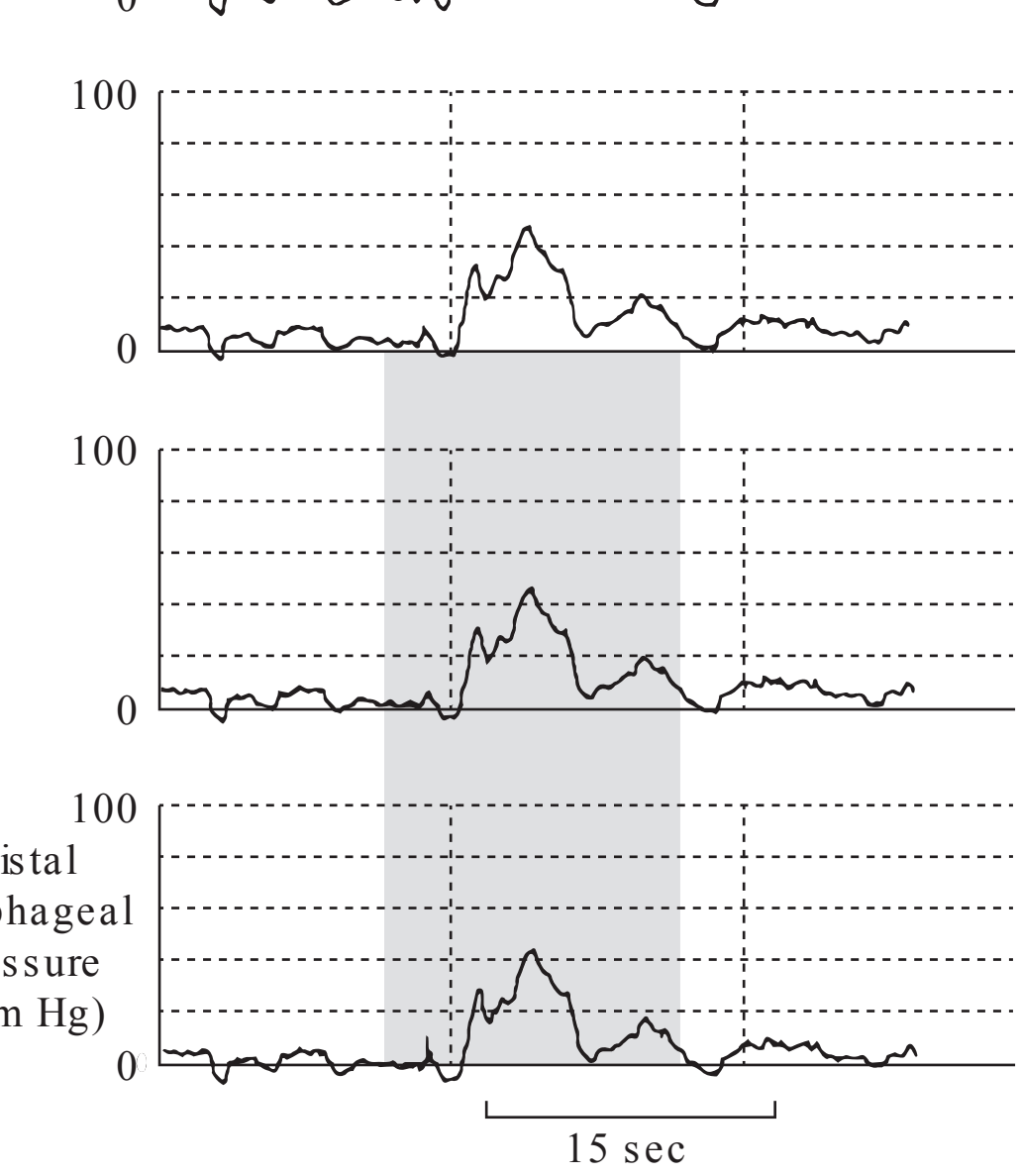
- EGD to rule out structural disorders beyond achala obstruction) that may present similarly to achalasia especially cancer.
- **Barium swallow (initial test):** Esophageal **dilation w tapering** of the distal esophagus (see Figure 2.6-2B).
- **Manometry (definitive test): ↑ resting LES pressur relaxation** upon swallowing, and **↓ peristalsis** in the gus (see Figure 2.6-3).

### Treatment

- **Short term:** Nitrates, calcium channel blockers, or e of botulinum toxin into the LES.
- **Long term:** Pneumatic balloon dilation or surgical (He

## ESOPHAGEAL DIVERTICULA

- Diverticula can be present in any location. **Zenk** defined as cervical outpouching through the cricopha is a posterior, false diverticulum.
- **Hx/PE:** Chest pain, dysphagia, halitosis, and regurgit food.
- **Dx:** Barium swallow will demonstrate outpouchings.
- **Tx:** If symptomatic, treat with surgical excision of th




**alasia.** Manometry with incomplete LES relaxation. (Reproduced with per-
F. Idiopathic (primary) achalasia, Orphanet J Rare Dis 2007 Sep 26;2:38.)

gia, initially to solids and later to liquids, is common. hagia, GERD, GI bleeding, and vomiting are also seen.

**nitial test):** Narrowing of esophagus with an irregular bor-
nto the lumen (see Figure 2.6-2C).
**sy:** Establishes the diagnosis.
**opic ultrasound:** Staging.

Esophag
because

SCC of t

for lifestyle modification and medical treatment).

- **EGD with biopsy:** Performed in patients whose symp
  tory to initial empiric therapy, (2) long standing (to ru
  agus and adenocarcinoma; see Figure 2.6-4), or (3) as
  **symptoms** (eg, blood in the stool, weight loss, dysp
  chest pain).
- **24-hour pH monitoring with impedance (definitiv**
  diagnosis is uncertain.
- **Other studies:** Indicated for refractory symptoms or
  causes. May include barium swallow (may demonstra
  2.6-2D) or esophageal manometry (see Figure 2.6-3).

### Treatment

- **Lifestyle:** Weight loss, head-of-bed elevation, small
  and avoidance of nocturnal meals and substances th
  avoid alcohol, chocolate, coffee).
- **Pharmacologic:**
  - **Mild/intermittent: Antacids.**
  - **Chronic/frequent: $H_2$ receptor antagonists** (cimet
    **PPIs** (omeprazole, lansoprazole).
  - Severe/erosive: PPIs first; if refractory, **Nissen fun**
    may be of benefit.
  - **Complications:** Erosive esophagitis, esophageal pe
    tion pneumonia, upper GI bleeding, **Barrett esoph**

## HIATAL HERNIA

- Herniation of stomach upward into the chest throu
  Three common types:
  - **Sliding hiatal hernia (95%):** Gastroesophageal jun
    of the stomach are displaced above the diaphragm
    atic (see Figure 2.6-5A).
  - **Paraesophageal hiatal hernia (5%):** Gastroes

MI.

nce of
rom a



**phagus**
n irreg-
action
ach)
e lower
n from
Medicine,




B

**tal hernia. (A) Sliding hiatal hernia. (B) Paraesophageal hiatal hernia.** (Reproduced w
osis & Treatment: Surgery, 13th ed. New York, NY: McGraw-Hill; 2010.)

## s of the Stomach and Duodenum

gastric mucosa. Subtypes:

Rapidly developing, superficial lesions, often due to
ohol, H pylori infection, and stress from severe illness (eg,
ry).
**s:**
**%):** Occurs in the fundus and is due to **autoantibodies to**
**.** Causes **pernicious anemia** and is associated with other
disorders and ↑ risk of gastric adenocarcinoma and carci-

**%):** Occurs in the antrum and may be caused by NSAIDs
**infection.** Often asymptomatic, but associated with an
ic ulcer disease (PUD) and gastric cancer. **Note:** H pylori
**but does not always cause gastritis.**

mptoms of epigastric pain, nausea, vomiting, hemateme-

Type A
pernicio
intrinsic
absorpt

Stress u
which a
injuries,
associat

| | |
|---|---|
| | for H pylori. |
| Hpylori on histology or culture. Can also detect l metaplasia, MALT, or widespread gastritis. | Gold standard for diagnosis of gas invasive test. |

- Give prophylactic PPIs to patients at risk for stre patients).

## GASTRIC CANCER

- Malignant tumor (mostly adenocarcinoma) with a po particularly common in Korea and Japan.
- **Risk factors:** Diet high in nitrites and salt and low (antioxidants), H pylori colonization, and chronic gastr
- **Hx/PE:** Early-stage disease is usually asymptomatic bu with indigestion and loss of appetite. Late-stage disease symptoms: abdominal pain, weight loss, and upper GI
- **Dx:** Upper endoscopy with biopsy (definitive test) to r gies and confirm the diagnosis.
- **Tx:** If detected early, treatment is surgical resection. M with late-stage, incurable disease. Five-year survival is disease.

## PEPTIC ULCER DISEASE

- Results from damage to the gastric or duodenal impaired mucosal defense and/or ↑ acidic gastric conte
- **Risk factors:** H pylori (> 90% of duodenal ulcers ulcers), **NSAIDs, alcohol,** and **tobacco use;** concom steroids and NSAIDs; **gender** (males > females).

**ent:**

**n suspected:** AXR (initial test) to rule out free air under m. Consider CT (definitive test) if AXR shows no perfora- e is high clinical suspicion. Then, surgery if perforation is CT. Carefully monitor BP.

**tive bleeding:** Serial hematocrits (initially), rectal vault NG lavage. Monitor BP and treat with IV hydration, sion, and IV PPIs. Then, perform an urgent EGD (defi- ol suspected bleeding.

**agement:**

**rapy goals:** Protect the mucosa, ↓ acid production, and ylori infection.

**ase:** Treat with antacids, PPIs, or $H_2$ blockers.

**infection:** Triple therapy (omeprazole, clarithromycin, icillin).

ue exacerbating agents (alcohol, tobacco).

**targeted biopsy:** Indicated in patients with symptoms lical therapy to rule out gastric cancer.

**y (eg, parietal cell vagotomy):** Severe cases refractory to

likely from posterior ulcers that erode into the gastroduo- outlet obstruction, perforation, intractable pain.

## ON SYNDROME

characterized by **gastrin-producing tumors** in the duode- creas that lead to oversecretion of gastrin.

gastrin leads to ↑ gastric acid production and recurrent or **s.**

gastrinomas are associated with multiple endocrine neo-

**FIGURE neum.** U under th mission fro

After a r is Greate Decreas

All gastr rule out

Misopro who req arthritis)

gap is 50–100 mOsm/kg).

$$\text{Stool osmotic gap} = 290 - 2 * (\text{stool Na} + \text{st}$$

### History/Pe

- **Acute diarrhea:** Acute onset with a duration of < 2 w
  tious and self-limited.
  - Multiple pathogens may be responsible (see Table
  - Common causes of **pediatric diarrhea** are **Rotavi**
    **and enterovirus infection.**
- **Chronic diarrhea:** Insidious onset with a duration of >
  - **Secretory:** Carcinoid tumors, VIPomas.
  - **Malabsorption/maldigestive/osmotic:** Bacterial
    atic insufficiency, mucosal damage, lactose intoler
    laxative abuse, postsurgical short bowel syndrome.
  - **Inflammatory/infectious:** IBD.
  - **Increased motility:** Irritable bowel syndrome (IBS)

### Diagnosis

- **Acute diarrhea:** No further studies are indicated unle
  high fever, bloody diarrhea, or diarrhea lasting > 4–5 d
- **Chronic diarrhea:** History/physical examination to na
  diagnosis. Additional studies include the following:
  - **Stool analysis:** Leukocytes, culture, C difficile toxi
    site exam (O&P).
  - **Sigmoidoscopy:** Uncommonly done, but usefu
    bloody diarrhea with no obvious clinical suspicion.

**TABLE 2.6-3. Types of Stool Osmotic Gap**

| Stool osmotic gap | description |
|---|---|

| | | |
|---|---|---|
| ood or water.<br>ects young children and oung adults; generally asts 7–10 days. | | |
| ociated with recent reatment with antibiotics penicillins, quinolones, **lindamycin**).<br>ects hospitalized adult atients.<br>**tch for toxic megacolon seen on AXR).** | Presents with fever, abdominal pain, and possible systemic toxicity. | Most commonly causes colitis, but can involve the small bowel.<br>Identify C diffici toxin in the stool.<br>Sigmoidoscopy shows pseudomembranes. |
| used by ingestion of ontaminated food or vater; look for a history f travel in developing ountries.<br>incubation period can ast up to 3 months. | Presents with severe abdominal pain and fever.<br>Endoscopy shows "flas - shaped" ulcers. | Chronic amebic colitis mimics IBD. |
| used by ingestion of ontaminated food (raw neat).<br>ects children and the lderly; generally lasts –10 days. | Presents with severe abdominal pain, low-grade fever, and vomiting. | It is important to rule out GI bleeding and ischemic colitis.<br>HUS is a potential complication, primarily in children. |
| used by ingestion of ontaminated poultry or ggs. | Presents with a prodromal headache, fever, myalgia, and abdominal pain. | Sepsis is a concern, as 5–10% of patients become bacteremic. |

- Inability to absorb macro- and/or micronutrients. C include the following:
  - **Mucosal abnormalities:** Celiac disease (associated petiformis; see Figure 2.6-7), Whipple disease, trop
  - **Bile salt deficiency:** Ileal disease in Crohn dise resections (> 100 cm of terminal ileum), bacterial c
  - **Other:** Pancreatic insufficiency, short bowel syndro
- **Hx/PE:** Presents with **frequent, loose, watery stools** (c sorption) and/or **pale, foul-smelling, bulky stools** (ste digestion) associated with abdominal pain, **flatus, bl nutritional deficiencies,** and fatigue.
- **Dx:** Multiple laboratory tests based on the clinical definitive.
- **Tx:** Etiology dependent. In severe cases, patients immunosuppressants, and anti-inflammatory medicatio

## LACTOSE INTOLERANCE

- Results from a **deficiency of lactase,** a brush-border lyzes lactose into glucose and galactose.
- Common among populations of African, Asian, an descent, also transiently after an acute episode of gastro
- **Hx/PE:** Presents with **abdominal bloating, flatulen watery diarrhea following milk ingestion.**
- **Dx:** Often treated empirically with lactose-free diet. **H** reveals ↑ hydrogen following the ingestion of lactose.
- **Tx:** Avoidance of dairy products; oral lactase enzyme re

## CARCINOID SYNDROME

- Due to the metastasis of **carcinoid tumors,** which m

and/or constipation, abdominal distention, and mucous
s often worsen with stress.
**toms:** Rarely awakens patients from sleep; vomiting, sig-
oss, and constitutional symptoms are uncommon.
sually unremarkable.

**Rome III diagnostic criteria):** At least 3 days in 3 months
minal discomfort that is (two or more criteria): (1) relieved
2) associated with a change in stool frequency or consis-
ted with a change in stool appearance.
**disorders** such as IBD using tests like CBC, TSH, elec-
ultures, abdominal films, barium contrast studies, and,
n biopsy.
nce of celiac disease in IBS, so celiac should be ruled out
S.

tients benefit from a strong patient-physician relationship.
d offer reassurance and should not dismiss the symptoms.
**plements** (psyllium); exclude gas-producing foods.
Symptomatic treatment with antispasmodics. TCAs and
Long-term medical therapy is usually not indicated.

## TRUCTION

ial or complete blockage of passage of bowel contents
ll bowel.
**esions** from prior abdominal surgery (60% of cases), **her-**
neoplasms (10–20%), intussusception, gallstone ileus,
BD, and volvulus.
ntinued passage of flatus, but no stool.

In the U
of SBO i
cause of

## Treatment

- **Initial management:** Fluid resuscitation.
- **Partial obstruction:** Supportive care may be sufficient NPO status, NG suction, IV hydration, correction of e ities, and Foley catheterization to monitor fluid status avoid opioids and anticholinergics (slow GI motility).
- **Complete obstruction:** Exploratory laparotomy ind SBO, ischemic necrosis, or refractory SBO symptoms.

# ILEUS

- Loss of peristalsis without structural obstruction.
- **Risk factors:** Recent surgery/GI procedures, severe me bility, hypokalemia or other electrolyte imbalances, h betes mellitus (DM), and medications that slow GI m linergics, opioids).

## History/Pe

- **History:** Diffuse, constant abdominal discomfort, **na** and an **absence of flatus or bowel movements.**
- **Examination:** Diffuse tenderness, **abdominal distenti bowel sounds; rectal examination required** to rule ou elderly patients.





porarily ↓ or discontinue oral feeds.
**ession:** Initiate **NG suction/parenteral feeds** as necessary.
: Replete electrolytes as needed; hydrate with IV fluids.

## EMIA

upply to the small intestine, resulting in ischemia and, . The most common causes are as follows:

boli most commonly originate in the heart. Risk factors rillation and stasis from a ↓ ejection fraction.
**cclusion from thrombosis:** Most commonly occurs in the The 1° risk factor is atherosclerosis.
onocclusive arterial disease (atherosclerosis of mesenteric r vasospasm), venous thrombosis (due to hypercoagulable state.

**abdominal pain out of proportion to the examination,** liarrhea, and bloody stools, prior episodes of abdominal ntestinal angina").

May reveal bowel wall edema ("thumbprinting") and air wall (pneumatosis intestinalis).
**angiography (definitive test):** Gold standard for diagnosis ive disease, but conventional angiography allows for inter- bosis/embolism.

resuscitation, broad-spectrum antibiotics.
**al thrombosis or embolism:** Anticoagulation and either

- **Diverticulitis:** Inflammation and, generally, micro-pe
  ticulum 2° to fecalith impaction.
- **Risk factors: Low-fiber and high-fat diet,** advanced
  those > 80 years of age), and connective tissue disorder

### History/Pe

- **Diverticulosis:** Often **asymptomatic** until patients p
  intermittent, painless bleeding, which can cause sy
  (fatigue, lightheadedness, dyspnea on exertion) when b
- **Diverticulitis: LLQ abdominal pain, fever,** nausea, a
  ration is a serious complication that presents with perit

### Diagnosis

- Clinical history is important to diagnosis.
- CBC may show **leukocytosis** or anemia.
- In diverticulitis, CT scan (used to diagnose) may rev
  abscess (see Figure 2.6-9).
- Colonoscopy provides the definitive diagnosis in diver
  ever, **avoid sigmoidoscopy/colonoscopy in patients w
  litis** in view of the risk of perforation.

ed in
in light



### Treatment

- **Uncomplicated diverticulosis:** Routine follow-up. En
  diet or fiber supplements.
- **Diverticular bleeding:** Bleeding usually stops spon
  and hydrate as needed. If bleeding does not stop, her
  copy, angiography with embolization, or **surgery** is ind
- **Diverticulitis:** Treat with **bowel rest** (NPO), NG
  severe), and **broad-spectrum antibiotics** (metronidaz
  nolone or a second- or third-generation cephalosporin
  initial stage.
- **For perforation:** immediate surgical resection of di

| | |
|---|---|
| **-pitched "tinkly" bowel sounds;** later, absence of wel sounds. | **High-pitched "tinkly" bow** bowel sounds. |
| **esions** (postsurgery), **hernias,** neoplasm, volvulus, susception, gallstone ileus, foreign body, Crohn disease, c fib osis (CF), stricture, hematoma. | **Colon cancer,** diverticulitis, benign tumors. **Assume colon cancer until** |
| paralytic ileus, gastroenteritis. | SBO, paralytic ileus, appendi (pseudo-obstruction). |
| electrolytes, lactic acid, AXR (see Figure 2.6-8); contrast es (determine if it is partial or complete), CT scan. | CBC, electrolytes, lactic acid, scan; water contrast enema sigmoidoscopy/colonoscop |
| italize. Partial SBO can be treated conservatively with **G decompression** and NPO status. nts with complete SBO should be managed gressively with NPO status, NG decompression, IV fluid , ctrolyte replacement, and **surgical correction.** | Hospitalize. Obstruction can enema, colonoscopy, or a is usually required. Ischem colectomy with a divertin Treat the underlying cause ( |

asymptomatic. When they are symptomatic, symptoms ion of the lesion:

**ons:** Often bulky, ulcerating masses that lead to **anemia ccult blood loss.** Patients may complain of weight loss, ea, weakness, or vague abdominal pain. Obstruction is n has a larger diameter than left colon.)

**s:** Typically **"apple-core" obstructing** masses (see Figure complain of **obstruction, change in bowel habits** (eg,

Iron def
patient
proven

| | |
|---|---|
| IBD | Ulcerative colitis > Crohn diseas |
| Adenomatous polyps | Villous > tubular; sessile > pedu |
| High-fat, low-fiber die | — |

el
a
n LBO
d colon
rance
of vol-
ssion from
Surgery,

### Treatment

- Surgical resection of the tumor is first-line treatment.
- **Adjuvant chemotherapy** is appropriate in cases with ⊕
- Follow with serial CEA levels to detect recurrence, CXR, and abdominal CT to screen for metastases.

## ISCHEMIC COLITIS

- Insufficient blood supply to the colon that results in is tially, necrosis. Most commonly affects the left col "watershed area" at the splenic flexure. Usually occu atherosclerosis.
- **Hx/PE:** Presents with crampy **lower abdominal pain diarrhea** after meals or exertion or in the heat. Fever suggest bowel necrosis.
- **Dx:**
  - **CT scan with contrast (initial test):** Thickened bo rosis.
  - **Colonoscopy:** Pale mucosa with petechial bleeding

**TABLE 2.6-7. Screening Recommendations for Colorectal Cancer**

| Risk Category | Recommendations |
|---|---|

atomic boundary between the duodenum and jejunum). ents the features of upper and lower GI bleeding.

## atory Bowel Diseas

**ease** (see Figure 2.6-12) and **ulcerative colitis** (see Figure non in Caucasians and **Ashkenazi Jews,** with onset most g in the teens to early 30s or in the 50s. Table 2.6-9 sum- s of IBD. In patients with a history of Crohn disease and in, suspect an SBO, which is due to the fistula formation. es not cause fistulas.



**FIGURE**
The enc
an "appl
descend
x-ray. (Re
Current Sur
ford, CT: Ap

**res of Upper and Lower GI Bleeding**

| upper gi bleeding | lower |
|---|---|
| atemesis ("coffee-ground" emesis), melena matochezia, hypovolemia (eg, tachycardia, headedness, hypotension). | Hematochezia > melena, bu |
| ube and lavage (may be ⊖ in 15% of upper GI bleeds); oscopy is definiti e. | Rule out upper GI hemorrha<br>Anoscopy/sigmoidoscopy fo<br>with small-volume bleedi<br>Colonoscopy if stable; arteri<br>laparotomy if unstable. |
| | |

**FIGURE 2.6-12. Crohn disease. (A)** Small bowel follow-through ( shows skip areas of narrowed small bowel with nodular mucosa (arrows) pare with normal small bowel (arrowhead). **(B)** Spot compression image "string sign" narrowing (arrow) due to stricture. **(C)** Deep ulcers in the c Crohn disease, seen at colonoscopy. (Image A reproduced with permission from Ch ed. New York, NY: McGraw-Hill; 2004. Image B reproduced with permission from USMLE-Rx.com permission from Fauci AS et al. Harrison's Principles of Internal Medicine, 17th ed. New York, NY: M

# Hernias

**Inguinal hernias are protrusions of abdominal content** intestine) into the inguinal region through a weakness or d inal wall. Classified as either direct or indirect. There ar **nias.** See Table 2.6-10 and Figure 2.6-14.

## Treatment

Because of the risk of **incarceration** and **strangulation,** s indicated.

# Biliary Disease

## CHOLELITHIASIS AND BILIARY COLIC

Colic results from transient cystic duct blockage from Although risk factors include the **four F's** (**F**emale, **F**at,







| | |
|---|---|
| urgency.<br>Examination may reveal orthostatic hypotension, tachycardia, abdominal tenderness, frank blood on rectal examination, and extraintestinal manifestations. | weight loss, watery dia<br>Examination may reveal f or mass, **perianal fissu** extraintestinal manifes |
| Aphthous stomatitis, episcleritis/uveitis, arthritis, **primary sclerosing cholangitis,** erythema nodosum, and pyoderma gangrenosum. | The same as ulcerative co skin, to the bladder, or be |
| CBC, AXR, stool cultures, O&P, stool assay for C diffici .<br>Colonoscopy can show diffuse and ontinuous rectal involvement, friability, edema, and **pseudopolyps.**<br>Definiti e diagnosis can be made with biopsy. | The same lab workup as t series with small bowel<br>Colonoscopy may show a ulcers, strictures, **"cobb** "Creeping fat" may also<br>Definiti e diagnosis can b |
| **5-ASA agents** (eg, sulfasalazine, mesalamine), topical or oral; corticosteroids for fla e-ups and immunomodulators (eg, azathioprine) or biologics (eg, Infliximа ) for refractory or moderate to severe disease.<br>**Total proctocolectomy can be curative** for long-standing or fulminant colitis or **toxic megacolon;** also ↓ cancer risk. | **Similar to UC: 5-ASA ag** ups. May require immu or biologics (eg, Inflixin to severe disease and f<br>Surgical resection may be perforation, stricture, fi |
| There is a **markedly ↑ risk of colorectal cancer** in long-standing cases (monitor with frequent fecal occult blood screening and yearly colonoscopy with multiple biopsies after 8 years of disease). | The incidence of 2° malig ulcerative colitis but is gr population. |

**es of Hernias**

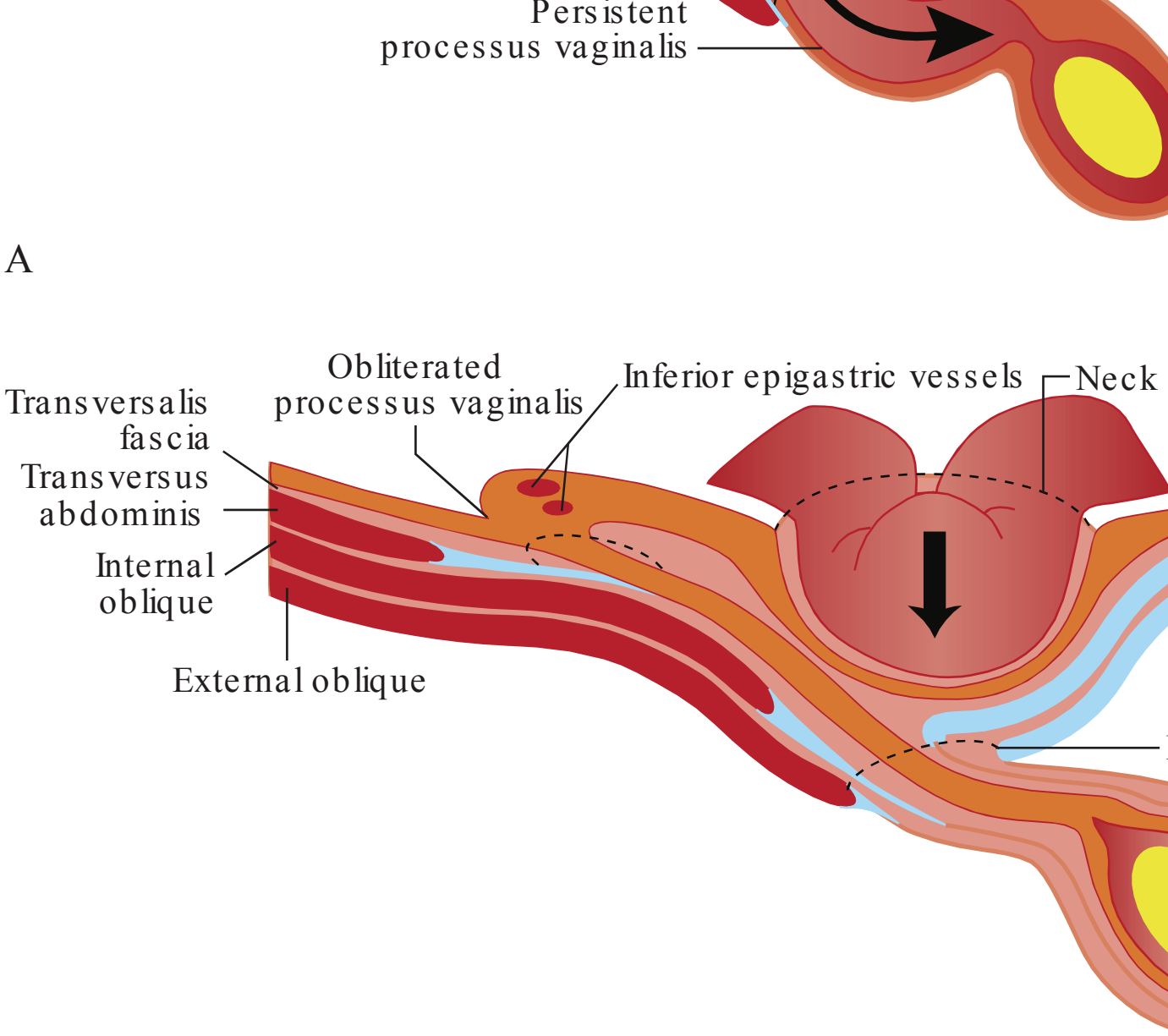


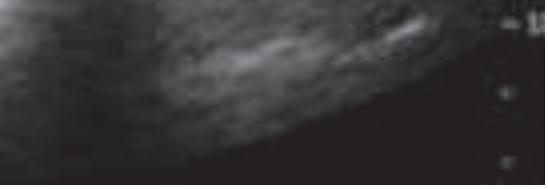

**FIGURE 2.6-14. Indirect (A) and direct (B) inguinal hernias.** (Repr
Tintinalli JE et al. Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 7th ed. New Yor

the disorder is common and can occur in any patient. A
include OCP use, rapid weight loss, chronic hemolysis
sickle cell disease), small bowel resection (loss of enteroh
bile), and TPN. Table 2.6-11 details the forms of biliary
the lab findings associated with each.

## History/Pe

- **Postprandial abdominal pain** (usually in the **RUQ**)

| | | | |
|---|---|---|---|
| | fatty meals, due to temporary occlusion of the cystic duct by a stone. | | |
| Inflamm tion of the gallbladder, typically due to stone occluding the cystic duct. | RUQ pain, fever (maybe), Murphy sign (cessation of inspiration with palpation of RUQ). | ↑ WBC, normal total bilirubin/alkaline phosphatase, amylase. | Ultrasonogra HIDA scan. |
| Stone in CBD. | Jaundice, ± RUQ pain, afebrile. | Normal/↑ WBC, ↑ total bilirubin/ alkaline phosphatase, ↑ amylase/lipase (if pancreatitis is present). | Ultrasonogra often does n the stone bu show dilated MRCP and E definiti e. |
| Infection of the CBD, usually due to stone in CBD. | Charcot triad: RUQ pain, fever, jaundice, Reynolds pentad: Charcot triad + shock and altered mental status. | ↑ WBC, ↑ total bilirubin/alkaline phosphatase. | Clinical diag confi med b dilation on i or ERCP (bot diagnostic a therapeutic) |





B

**disease. (A) Cholelithiasis.** Ultrasound image of the gallbladder shows a gallstone (a
**tis.** Ultrasound image shows a gallstone (red arrow), a thickened gallbladder wall (arr
iver. (Reproduced with permission from USMLE-Rx.com.)

### Diagnosis

- **Ultrasound (initial test):** May demonstrate stones, bil
  cystic fluid, a thickened gallbladder wall, gas in the wa
  and/or an ultrasonic Murphy sign (see Figure 2.6-15).
- When ultrasound is equivocal, obtain a HIDA scan. I
  icine scan, which uses a radiotracer excreted through
  nonvisualization of the gallbladder suggests acute chol

### Treatment

- Give broad-spectrum **IV antibiotics** and **IV fluids.**
- **Cholecystectomy** is indicated.

## CHOLEDOCHOLITHIASIS

- **Gallstones in the CBD.** Symptoms vary accordin
  obstruction, the duration of the obstruction, and the
  infection.
- **Hx/PE:** Biliary colic, jaundice, afebrile unless curre
  pancreatitis.
- **Dx: ↑ alkaline phosphatase** and **total and direct bilir**
  11).
- **Tx:** Management consists of ERCP with sphincteroto
  lecystectomy.

in,
lds
ndice,
ental

via ERCP/sphincterotomy, percutaneous transhepatic
n decompression.

truction resulting from the passage of a large (> 2.5 cm)
owel through a cholecystoduodenal fistula. Obstruction is
ecal valve.
ssic presentation is a subacute SBO in an elderly woman.
e no history of biliary colic.
haracteristics of SBO and pneumobilia (gas in the biliary
e diagnosis. Upper GI barium contrast images will dem-
ast in the colon.
with stone extraction; closure of the fistula and cholecys-

ease

FUNCTION TESTS

e divided into several patterns based on LFT results:

**njury:** ↑ AST and ALT.
**kaline phosphatase and bilirubin.**
**ation of hepatocellular and cholestatic picture.**
**lirubinemia:** ↑ bilirubin.

nical sign that occurs when bilirubin levels exceed 2.5 mg/
nd 2.6-17 summarize the clinical approaches toward cho-
hyperbilirubinemia.

HCV is C
with HC
hepatiti

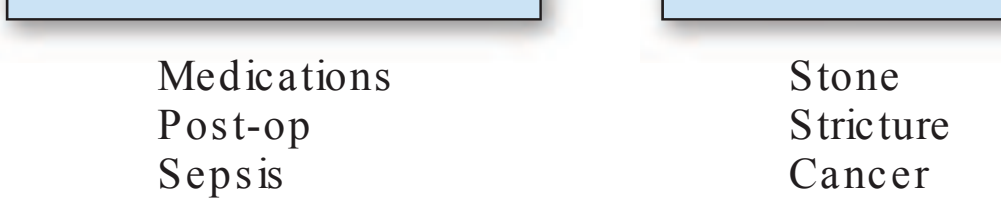



**FIGURE 2.6-16. Approach to cholestasis.**

## History/Pe

- **Acute hepatitis:**
  - Often begins with a nonspecific viral prodrome (
    pain, fatigue, URI symptoms, **nausea, vomiting,**
    habits) followed by **jaundice** and RUQ tenderness
    reveals jaundice, scleral icterus, and tender hepator
  - HAV and HEV have only a self-limited acute pha
    may feature a mild acute phase or none at all. Ace
    can cause a life-threatening hepatitis. Table 2.6-12
    tinctions among these.
- **Chronic hepatitis:** May be asymptomatic, but may cau
  and muscle pains. Jaundice and complications of
  (hepatic encephalopathy, ascites, esophageal varices)
  when the disease progresses to cirrhosis. At least 80
  with HCV and 10% of those with HBV in adulthood
  tent infection with chronic active hepatitis.

## Diagnosis

- **Acute hepatitis:** Labs reveal markedly ↑ **ALT and AS**
  alkaline phosphatase.
- **Chronic hepatitis:** ALT and AST are either mildly el
  mal/low for > 3–6 months. Contrast this with the n
  acute hepatitis as above.

Isolated hyperbilirubinemia (↑↑ bi

| | | |
|---|---|---|
| | treatment with antivirals or liver transplant in severe cases. | |
| fluid | Asymptomatic or viral prodrome and/or jaundice. | 80% become chro transmitted than acute phase. Very |
| fluid | Co-infection with HBV or superinfection in patient with prior HBV (more severe). | Requires HBV surf on HBV. |
| oral | Typically self-limited acute hepatitis similar to HAV. | High mortality rat become chronic i |

l hepatitis is made by **hepatitis serology** (see Table 2.6-13 8 for a description and timing of serologic markers). May psy if diagnosis is uncertain or to rule out other causes of hronic or severe cases.
studies include the following:
e **hepatitis:** ⊕ anti-nuclear and anti–smooth muscle anti- 1) and anti-liver-kidney microsomal-1 antibodies and anti-

**Hepatitis Serologic Markers**

| | d eScr ipt io n |
|---|---|
| | antibody to HAV; the best test to detect acute HAV. |
| | gen found on the surface of HBV; continued presence indicates carrier e. |

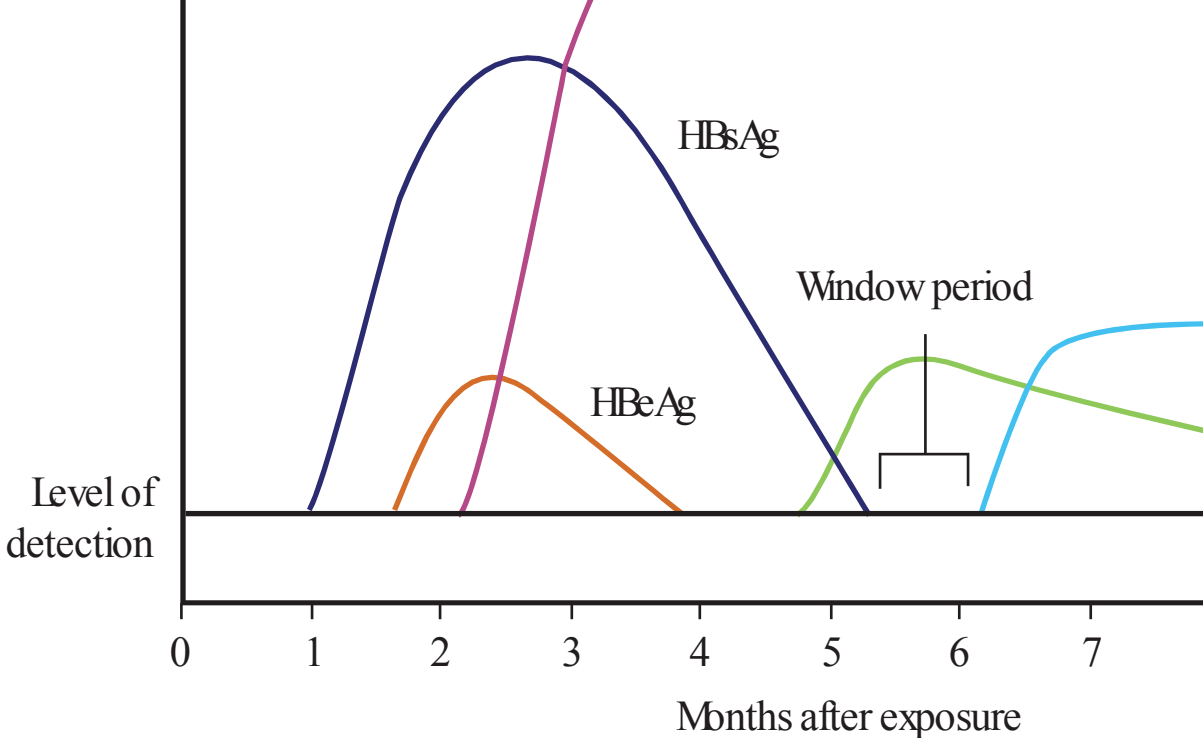



**FIGURE 2.6-18. Time course of hepatitis B with serologic marke**

sion from Le T et al. First Aid for the USMLE Step 1 2015. New York, NY: McGraw-Hill; 2015.)

liver cytosol antibodies (type 2). May also present
gamma globulins (IgG) and p-ANCA.

- **Hemochromatosis:** ↑ ferritin and transferrin satu
  biopsy showing high hepatic iron index.
- **Wilson disease:** ↓ ceruloplasmin, ↑ urine copp
  rings. Liver biopsy if diagnosis is uncertain.

## Treatment

- **Acute hepatitis:** Generally supportive care. Acute HB
  ment with antivirals.
- **Chronic hepatitis:** Treatment is etiology specific.
  - **Chronic HBV infection:** Tenofovir and entecavir
    used and have the least viral resistance. Other age
    dine, adefovir, and lamivudine, but they are not re
    high rate of resistance.
  - **Chronic HCV infection: Medications and treat**
    **based on genotype, cirrhosis status, and history**

ic causes include right-sided heart failure, constrictive
is, and Budd-Chiari syndrome (hepatic vein thrombosis
rcoagulability).

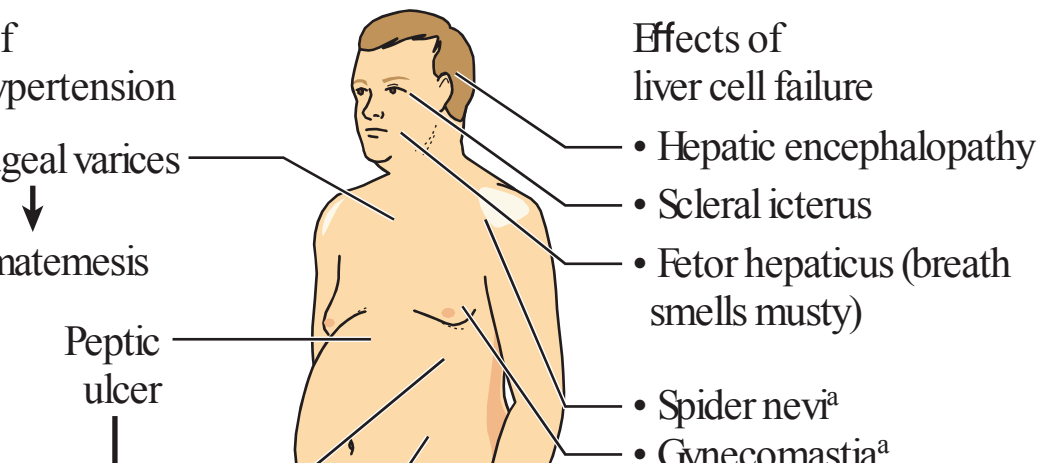
**ymptomatic,** though may present with **jaundice, easy**
**opathy),** and **complications of portal hypertension such**
**tic encephalopathy** (asterixis, altered mental status), **gas-**
**arices,** and thrombocytopenia. Ascites can be complicated
**bacterial peritonitis.**
an enlarged, palpable, or firm liver and other signs of por-
and liver failure (see Figures 2.6-19 and 2.6-20).

Spontan
diagnos
ascitic fl

**nction (initial tests): ↓ albumin, ↑ PT/INR,** and ↑ **biliru-**

**sion:** Thrombocytopenia (2° to hypersplenism, sequestra-
in the liver, and ↓ thrombopoietin production), varices,
esis).
**ve):** Biopsy will show bridging fibrosis and nodular regen-

tis serologies, autoimmune markers, serum ferritin, ceru-
$_1$-antitrypsin.



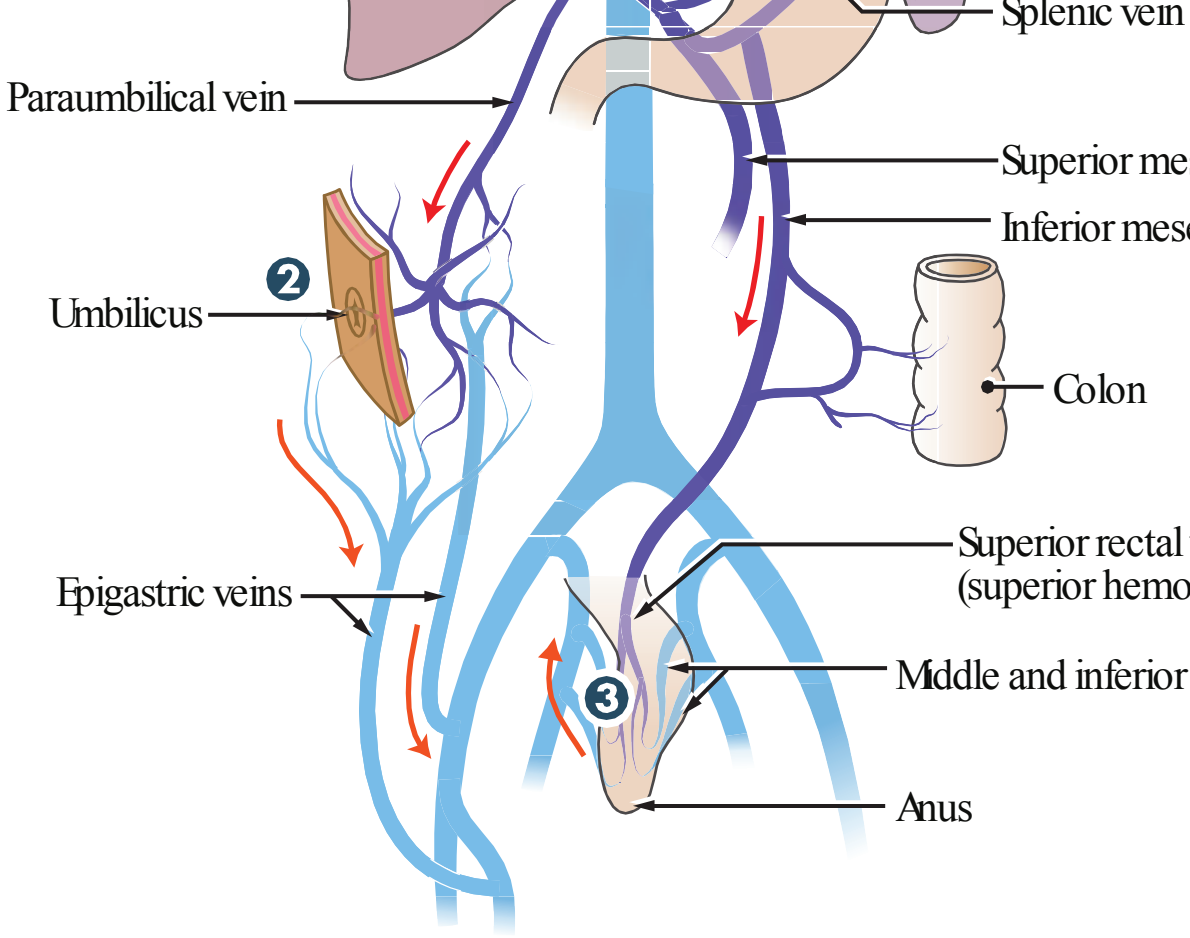



**FIGURE 2.6-20. Portosystemic anastomoses.** 1. Left gastric–azyg
ces. 2. Superior–middle/inferior rectal → hemorrhoids. 3. Periumbilica
caput medusae (navel). 4. Gastrorenal-splenorenal. 5. Retroperitoneal
with permission from Le T et al. First Aid for the USMLE Step 1 2015. New York, NY: McGraw-Hill

- If ascites is present, the etiology of ascites can be **serum-ascites albumin gradient** (**SAAG** = serum albu min); see Table 2.6-14.

## Tr ea Tmen T

The goal is to treat and prevent the progression of cirrhosi tors that can lead to decompensation (see Table 2.6-15).

**TABLE 2.6-14. Etiologies of Ascites by SAAG**

| Saag > 1.1 | Saag |
|---|---|

| | | |
|---|---|---|
| | Treatment indicated if diagnostic paracentesis reveals > 250 PMNs/mL. | prevent recurrence. D<br>with poor 1-year prog |
| e | Acute prerenal failure in the setting of severe liver disease. Due to splanchnic vasodilation and decreased blood fl w to the kidneys. A diagnosis of exclusion. Urinary sodium is < 10 mEq/L. "Healthy kidneys in an unhealthy environment." | Initially trial of volume<br>causes of renal failure.<br>splanchnic vasodilatio<br>blood pressure). May<br>Liver transplantation |
| hy | ↓ clearance of ammonia; often precipitated by dehydration, infection, electrolyte abnormalities, and GI bleeding. | Lactulose, and/or rifax<br>triggers. |
| | Portal hypertension leads to ↑ f ow through portosystemic anastomoses. | Endoscopic surveillan<br>medical prophylaxis<br>band ligation to pre<br>known varices.<br>For acute bleeding, en<br>sclerotherapy is ind<br>cases (associated w |
| | Impaired synthesis of all clotting factors (except VIII). | For acute bleeding, ad<br>**Vitamin K will not co** |

## ING CHOLANGITIS

isorder characterized by progressive inflammation and anied by strictures of extrahepatic and intrahepatic bile se usually presents in **young men with ulcerative colitis.**

## NON-ALCOHOLIC FATTY LIVER DISEASE

- Steatosis of hepatocytes leads to liver injury. Some pati
  alcohol steatohepatitis (NASH) and are at risk of liver fi
- Associated with insulin resistance and the metabolic sy
- **Dx:** Largely a diagnosis of exclusion. Liver biopsy m
  steatohepatitis.
- **Tx:** Weight loss, diet, and exercise. If non-alcoholic st
  ent, consider vitamin E and pioglitazone.

## HEPATOCELLULAR CARCINOMA

- One of the most common cancers worldwide despite it
  dence in the United States.
- **Risk factors:** In the United States risk factors are **cirr**
  HCV, and non-alcoholic steatohepatitis) and **chroni**
  **without cirrhosis).** In developing countries, **HBV infe**
  (in various food sources) are major risk factors.

### History/Pe

- Patients commonly present with **RUQ tenderness, ab**
  and signs of chronic liver disease such as **jaundice, ea**
  **coagulopathy.** May present as decompensation of prev
  cirrhosis.
- Examination may reveal tender **enlargement** of the liv

### Diagnosis

Often suggested by the presence of a mass on **ultrasound**
abnormal LFTs and significantly elevated α-fetoprotein
is required if diagnosis is uncertain.

ay present with abdominal pain, **DM, hypogonadism, ne MCP joints, heart failure,** impotence, or cirrhosis.
**ronze skin pigmentation, cardiac dysfunction** (CHF), nd testicular atrophy. Labs may reveal evidence of DM.
is does not affect the lung, kidney, or eye.

ercent saturation of iron, and ferritin with ↓ serum trans-

ration (serum iron divided by TIBC) > 45% is highly sug-
erload.
**biopsy** (to determine hepatic iron index), MRI, or HFE creen.

**tomy** to normalize serum iron levels, and then mainte-
ny every 2–4 months.
gents such as **deferoxamine,** deferiprone, or deferasirox maintenance therapy.

**ellular carcinoma,** restrictive cardiomyopathy, arrhyth-
ce, arthropathy, hypopituitarism.

## HEPATOLENTICULAR DEGENERATION)

cessive disorder that results in defective copper transport accumulation and deposition of copper in the liver and ccurs in patients < 30 years of age; 50% are symptomatic

resent with hepatitis/cirrhosis, neurologic dysfunction



**FIGURE ring.** No iris. This in Desce finding i
permission

symptoms with correction of hypoglycemia.

- **Dx:**
  - **Lab tests (initial tests):** Fasting serum insulin (e (elevated).
  - **72-hour fasting (definitive test):** Patient develops tomatic hypoglycemia after prolonged fast. Onc reached, labs drawn to determine etiology: glucose, (elevated), C-peptide level (elevated), sulfonylure serum beta hydroxybutyrate level (low), serum co elevated).
  - **Tumor localization:** transabdominal ultrasound scopic ultrasound, or selective arterial calcium stim
- **Tx:** Surgery to resect tumor.

## VIPOMA

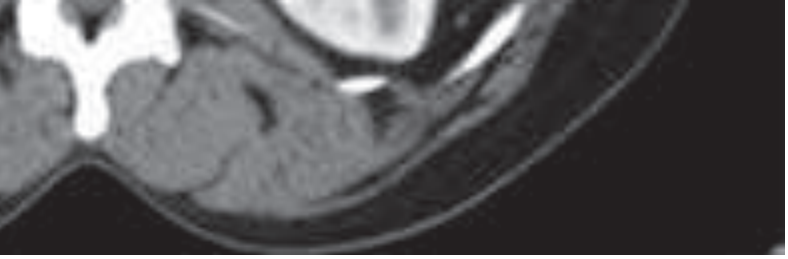
- Results from VIP-producing tumor, highly malignant.
- **Hx:** Watery diarrhea, dehydration, muscle weakness, fl
- **Dx:**
  - **Stool sample:** Low stool osmotic gap (ie, secretory
  - **Lab tests:** High VIP levels, achlorhydria (because inhibited by VIP), hyperglycemia, hypercalcemia, h
  - **CT scan:** Localize the tumor.
- **Tx:** Initially, replace fluid and electrolyte losses. Surg May also consider octreotide.

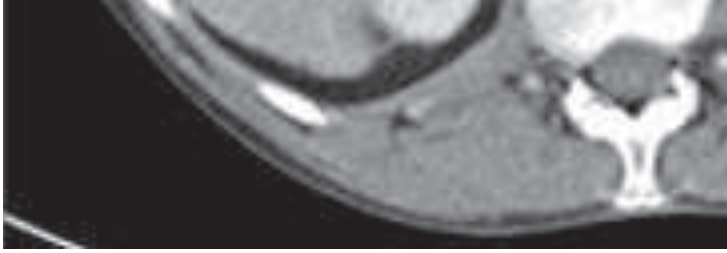
## PANCREATITIS

Table 2.6-16 outlines the features of acute and chronic pa

## PANCREATIC CANCER

| | |
|---|---|
| acute pancreatitis in children), drug side effects (thiazide diuretics), viral infections, post-ERCP, scorpion bites. | |
| **Severe epigastric pain (radiating to the back);** nausea, vomiting, weakness, fever, shock, pleural effusion , ARDS. Flank bruising **(Grey Turner sign)** and periumbilical discoloration **(Cullen sign)** may be evident on examination. | Recurrent episodes of **pe** anorexia, nausea, constip weight loss, DM. |
| ↑ **lipase (more sensitive and specific than a ylase),** ↑ **amylase,** ↓ **calcium** if severe; **"sentinel loop"** or **"colon cutoff sig "** on AXR.<br>Abdominal ultrasound or CT may show an enlarged pancreas with stranding, abscess, hemorrhage, necrosis, or pseudocyst (see Figure 2.6-22A). | ↑ to normal amylase and **pancreatic calcifi ation** alternating stenosis and d duct on CT or US **("chain** |
| Removal of the offending agent if possible.<br>Supportive care, including IV fluids/ele trolyte replacement, analgesia, bowel rest, NG suction, nutritional support, and $O_2$.<br>Infected pancreatic necrosis should be treated with antibiotics, though prophylactic antibiotics are not recommended. Endoscopic, percutaneous, or surgical debridement may be considered. | Analgesia, pancreatic enz causative agents (EtOH), dilation of pancreatic duc structural causes. |
| Roughly 85–90% are mild and self-limited; 10–15% are severe, requiring ICU admission. Mortality may approach 50% in severe cases. | Patients can have chronic dysfunction. |
| **Pancreatic pseudocyst, fistula ormation,** hypocalcemia, renal failure, pleural effusion, ch onic | **Chronic pain,** opiate add malnutrition/weight loss |





B

**is. (A) Uncomplicated acute pancreatitis.** Peripancreatic fluid and fat stranding can
**tis.** Note the dilated pancreatic duct (arrowhead) and pancreatic calcifications (arrow

## Treatment

- **Locally advanced or metastatic disease:** Most freque
  liative chemotherapy or best supportive care.
- **Small tumors in the pancreatic head with no metast**
  **involvement:** Whipple procedure (pancreaticoduoden
- Chemotherapy with 5-FU and gemcitabine may imp
  vival, but long-term prognosis is poor (5–10% 5-year su
- **ERCP with stenting** to relieve patients presenting wi
  toms.

168 White Blood Cell Disorders
168 Leukemias
168 Lymphomas
169
Plasma Cell Disorders
170
Multiple Myeloma
172 Waldenström Macroglobulinemia
172 Amyloidosis
173
Neutropenia
173
Eosinophilia
173
180
Transplant Medicine
180
182 Diseases Associated With Neopla
182


ULAR Coagulation

Idiopathic Thrombocytopenic Purpura

Thrombocytopenic Purpura

ers

ation

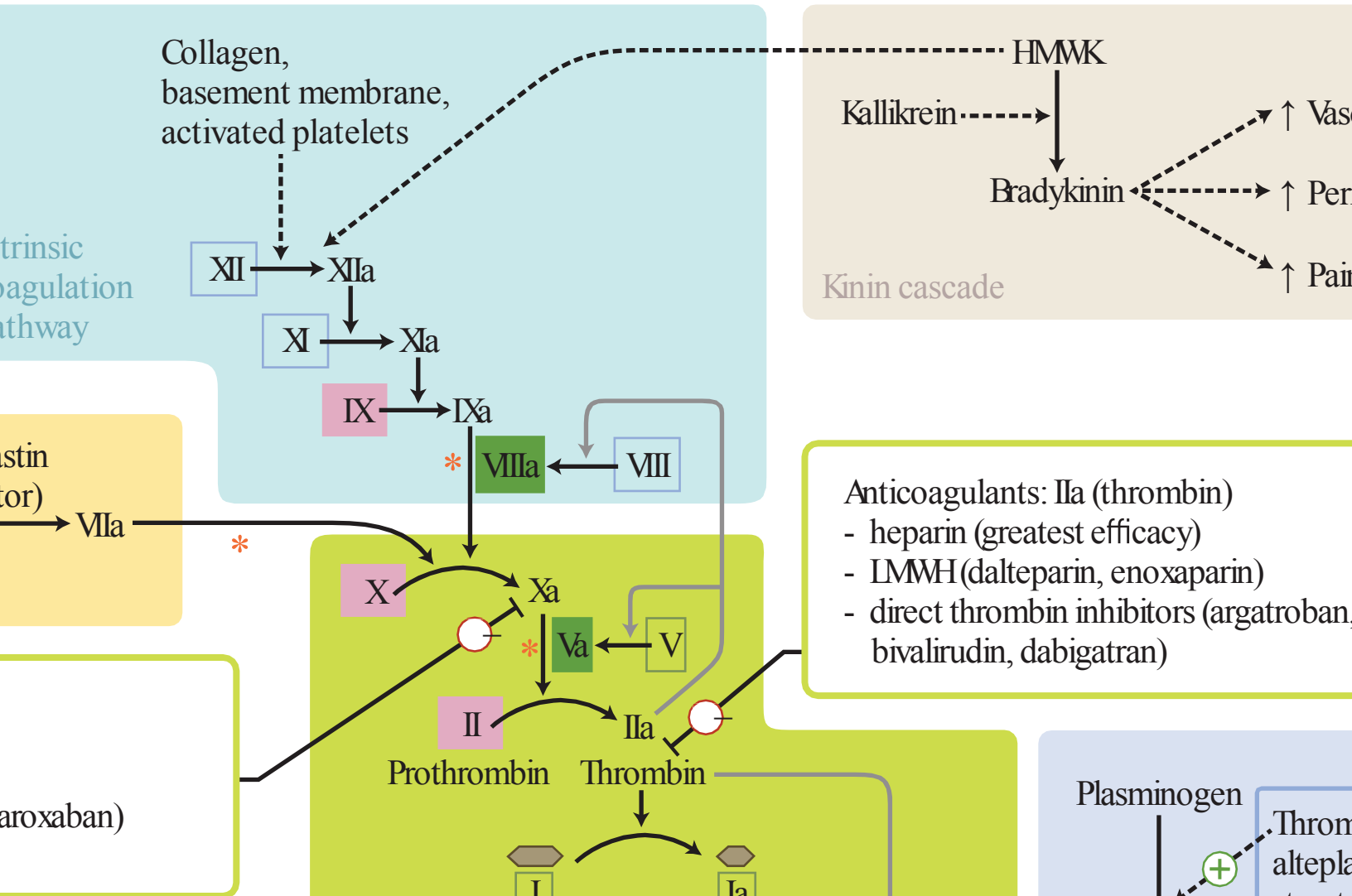
ations

ate a **fibrin mesh,** as shown in Figure 2.7-1. Drugs affec
shown in Table 2.7-1.

Heparin-to-warfarin bridge is necessary because prot
shorter half-lives than the other vitamin K–dependent
and X), leading to a transient period of **paradoxic hyper**
proper anticoagulation.

## HEMOPHILIA

**X-linked clotting factor deficiencies** of factors **VIII** (her
**cases**), **IX** (hemophilia **B**), and **XI** (hemophilia **C**) that
**dency to bleed.** Rarely, hemophilias can be **acquired** i
these factors are produced via autoimmune, lymphoprol
tum states.



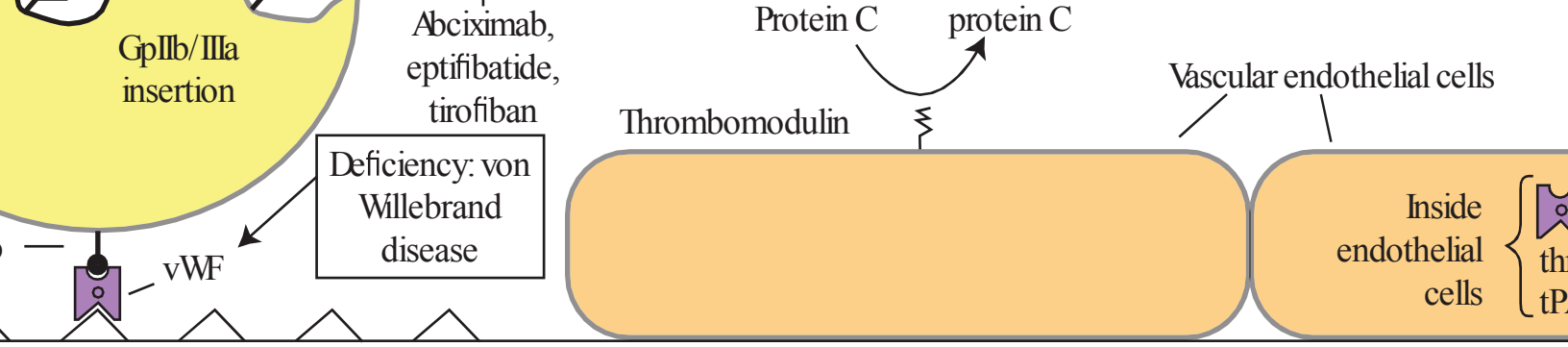
| | | | |
|---|---|---|---|
| | VII, IX, X, proteins C and S). | | Te |
| | Aid conversion of plasminogen to plasmin, which breaks down fib in. | ↑ **PT,** ↑ **PTT.** | Tr ac |
| pan) | Directly inhibit factor Xa. | **PT/PTT not monitored.** | **N** |
| | Mainly inhibits factor Xa. | **Anti-factor Xa,** although typically unmonitored. | Pr th |
| itors an) | Directly inhibits factor II (thrombin). | | N |

ung boy with **spontaneous hemorrhage** into the tissues if untreated, can lead to **arthropathy and joint destruc-** us **intracerebral,** renal, retroperitoneal, and GI hemor- een.
have major hemorrhage after surgery, trauma, or dental re otherwise asymptomatic.

ged on basic bleeding workup. PT and bleeding time are

**Mixing study.** Mix the patient's plasma with normal vill **correct the PTT** in hemophilia patients as it contains rs.
est: **Obtain specific factor assays** for factors VII, VIII, IX,

Cryopre
factor V
concent
fibronec




**hesis deficiencies.** (Adapted with permission from Le T et al. First Aid for the USMLE Step 1 2015. New York, NY

due to platelet dysfunction and deficient factor VIII, and hemophilia. vWD is the **most common inherited bleed** the population), and the most common form of vWD is t moderate deficiency in vWD). Type 2 includes qualitati and type 3 is absence of functional vWF.

### History/Pe

- Often presents in childhood with **recurrent and** bleeding (epistaxis, gums, teeth, menorrhagia) and ble surgical procedures.
- Often a family history is present, and symptoms worsen

### Diagnosis

- Initial bleeding workup will show an ↑ **bleeding tim** ↑ PTT as in hemophilia may be seen due to low factor **platelet count will be normal.**
- Most accurate test: **Ristocetin cofactor assay** of patien capacity of vWF to agglutinate platelets) and **vWF anti**

### Treatment

- Bleeding episodes in mild/moderate type 1 disease **DDAVP.**
- Menorrhagia can be controlled with **OCPs.** Avoid AS

| | |
|---|---|
| y | Antiphospholipid syndrome |
| A mutation | Nephrotic syndrome |
| n | OCPs/HRT |

**n:** A polymorphism in factor V, rendering it resistant to ctivated protein C, is the most common cause of heredi- polic disease.

**d thrombocytopenia (HIT):** An immunologic reaction to on of heparin creates platelet-activating antibodies, which nation of blood clots and a **rapid (50%) drop in platelet**

**id syndrome (APS):** Often associated with **SLE** and **rheu-** APS predisposes to both arterial and venous thrombi for- taneous abortions.

n **recurrent** thrombotic complications: **DVT, pulmonary ial thrombosis, MI, and stroke.** Women may have recur- s.

d patient on anticoagulation with a drop in platelets.

: Young Caucasian patient with a personal and family his- is.

d women with recurrent miscarriages or thrombosis.

ary workup is conducted, acquired causes should be ruled abnormality is **confirmed with two abnormal values the patient is asymptomatic and untreated, with similar in two other family members.**

confirm HIT (PF4 antibody and serotonin release assay), oagulant and anticardiolipin antibody).

Protein
hyperco
**tissue**
**admini**

Suspect
onset of
hypoxia
arterial
another

ARDS.

- Clinical presentation:
  - **Acute:** Bleeding from venipuncture sites, into orga and petechiae.
  - **Chronic:** Bruising and mucosal bleeding, thrombo function, and transient neurologic syndromes.

### Diagnosis

- Diagnosed by clinical features and lab findings.
- DIC may be confused with liver disease, but **unlike VIII is depressed.**

### Treatment

**Reverse the underlying cause;** transfuse RBCs, platelets shock.

## THROMBOTIC THROMBOCYTOPENIC PURPURA

A deficiency of the vWF-cleaving enzyme **(ADAMTS-13)** mally large vWF multimers that aggregate platelets and cr **thrombi.** These block off small blood vessels, leading to **RBCs are fragmented** by contact with the microthromb lysis (microangiopathic hemolytic anemia). The ADAM either inherited or often acquired secondary to infection.

### History/Pe

Maintain high clinical suspicion for TTP if three of thes present:

- Low platelet count
- Microangiopathic hemolytic anemia
- Neurologic changes (delirium, seizure, stroke)

tes in the marrow. The **most common immunologic disor-**
**ildbearing age.**

el well with no systemic symptoms. They may have minor
bleeding, easy bruising, petechiae, hematuria, or melena.
s no splenomegaly.
with a range of conditions, including **lymphoma, leuke-**
**infection, and HCV infection.** Can present acutely or as

t onset of hemorrhagic complications following a viral ill-
only affects children 2–6 years of age, with boys and girls
lly.
idious onset of symptoms or incidental thrombocytopenia
roid-binding globulin (CBC). Affects adults 20–40 years of
> men.

**xclusion:** Once other causes of thrombocytopenia have
diagnose by Hx and PE, a CBC, and a peripheral blood
ormal RBC morphology.
opsy would show ↑ megakaryocytes but is done only in
patients > 60 years of age.

30,000 and no bleeding: **No treatment required.**
30,000 or clinically significant bleeding symptoms: **Corti-**
**IG.**
fails to improve or bleeding recurs, consider **splenectomy**
rombopoietin (TPO) receptor agonist to increase platelet
**iplostim** or **eltrombopag**).
ents, severe thrombocytopenia may occur in the fetus.

**FIGURE**
fragmen
in micro
and in m
permission
of Hemato

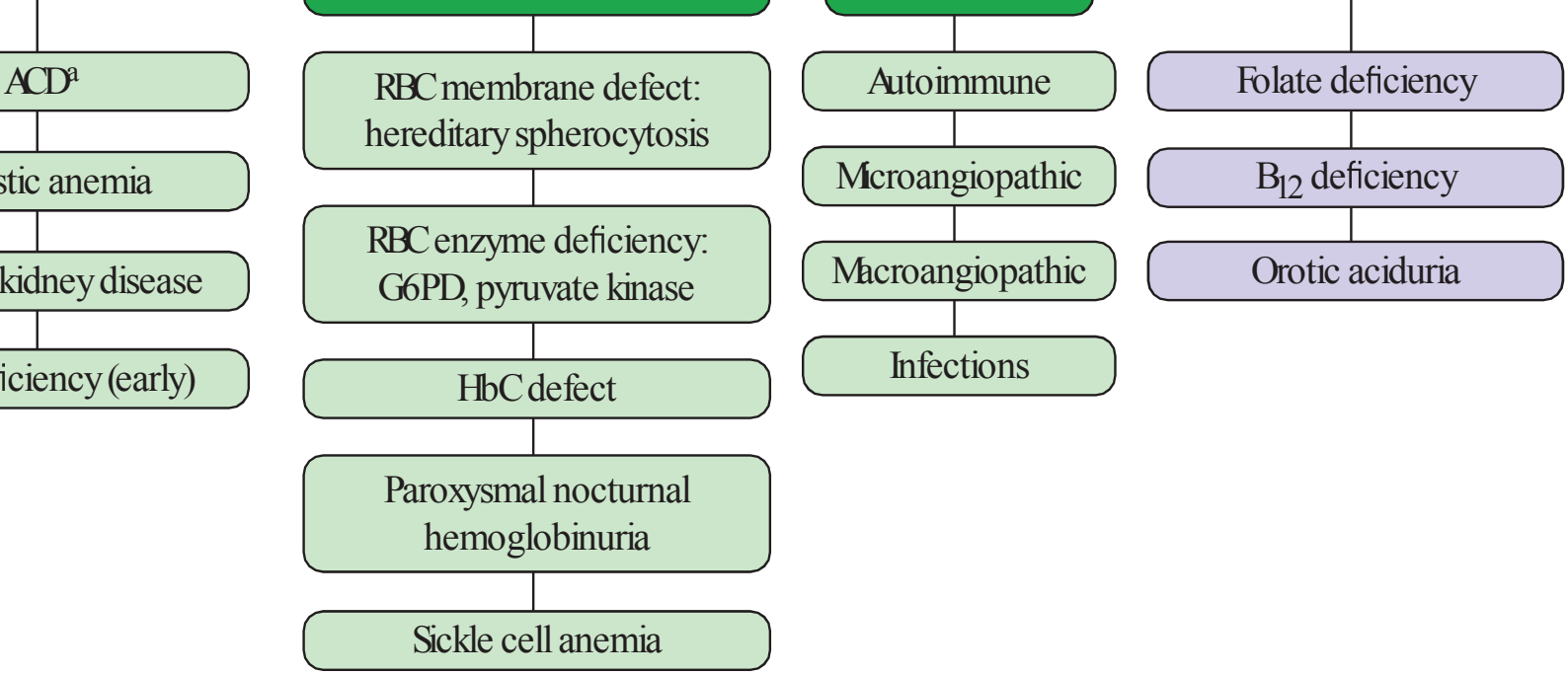
Differe
thromb

**HIT SH**

**H**IT or **H**
**I**TP
**T**TP or **T**
**S**plenom
**H**eredita
**O**ther ca
**C**hemoth

Anti-D (
rituxima

Anti-D (
as "deco




esent as a normocytic anemia and then progress to a microcytic anemia.
deroblastic anemia.

**rithm.** ACD = anemia of chronic disease; HbC = hemoglobin C. (Reproduced with perm
Graw-Hill; 2015.)

erly
l cancer
d to rule

## Iron Deficiency Anemia (a Microcytic Anemia

Anemia in which iron loss exceeds intake. May occur (growth phase, pregnancy, EPO therapy) or ↓ **iron** (chron bleeding, malnutrition/absorption disorders like celiac). **T girls,** and **women of childbearing age** are most commonl

### History/Pe

- Symptoms: **Fatigue, dyspnea, tachycardia,** and **pica.**
- If the anemia develops slowly, patients are generally as
- Physical findings: **Glossitis, conjunctival pallor, c onychia** (**"spoon nails,"** see Figure 2.7-5).

### Diagnosis

- Best initial test is a **CBC with iron studies** (see Tab value will make the diagnosis, but the constellation

| | | | |
|---|---|---|---|
| ptor | ↑ | Normal | Normal/↑ |
| | ↑ | Normal | Normal/↑ |

can be administered but is associated with a small risk of cts, including **anaphylaxis.** Iron sucrose may have a lower

## Inflammation/Diseas

proliferation, the body "hides" its iron in situations of on such as infection, malignancy, RA, or SLE. This results mocytic anemia with **high levels of iron storage in the** low serum iron (see Table 2.7-3).

## ia

a due to defects in heme metabolism that can be heredi- ommon causes include **alcohol, lead poisoning, chlor-** and malignancy). It presents with a low hemoglobin, **a,** basophilic stippling (in lead poisoning), and ringed sid- (see Figure 2.7-7).

## mia (a Macrocytic Anemia)

thesis secondary to **vitamin $B_{12}$ (cobalamin)** or **folate** megaloblastic anemia. $B_{12}$ deficiency is due to intestinal itionally from **pernicious anemia** (autoimmune destruc- , which produce the intrinsic factor needed for cobalamin lietary intake (vegetarians). Folate deficiency results from tary folate, or malabsorption. **Drugs that interfere with** luding chemotherapeutic agents (methotrexate, 6-mercap- to megaloblastic anemia.



**FIGURE**
**mia.** No
RBCs ("
areas of
permission
of Hemato



A

**FIGURE**
**RBC for**
**(B)** Ring
with permi
of memora
December
Paulo Henr

properly absorbed, as the liver's $B_{12}$ receptors will be sa
dose.

- **Radiolabeled $B_{12}$ in urine:** Dietary $B_{12}$ deficiency.
- **No radiolabeled $B_{12}$ in urine:** Consider perniciou
overgrowth, or pancreatic enzyme deficiency; test
the addition of intrinsic factor, antibiotics, or pan
radiolabeled $B_{12}$.

### Treatment

Correct the underlying cause of the anemia.

## Hemolytic Anemia (a Normocytic Anemia)

Occurs when bone marrow production is unable to comp
tion of circulating blood cells. Etiologies include the follo

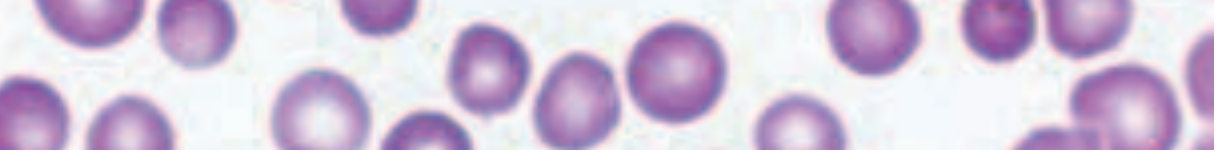
- **Glucose 6-phosphate dehydrogenase (G6PD) defi
nocturnal hemoglobinuria (PNH), spherocytosis** (H
**cell disease,** and **autoimmune anemia** will be discuss
but the general principles in this section will apply.
- **Microangiopathic hemolytic anemia:** TTP, HUS, DI
- **Mechanical hemolysis:** Associated with **mechanical h**
- **Other:** Malaria, hypersplenism.

### History/Pe

- Present with **pallor, fatigue, tachycardia,** and **tachypn**
- Patients are often **jaundiced,** with ↑ reticulocyte coun
and LDH, and a ↓ haptoglobin. Urine is dark with **h**
there is ↑ excretion of urinary and fecal urobilinogen.

### Diagnosis

Clinical. ↑ **LDH,** ↑ **indirect bilirubin,** ↑ **reticulocyte c
globin** are commonly seen.



**erocytes.** These RBCs (arrows) lack areas of central pallor. Spherocytes are found in autoimmune hemolysis and in hereditary spherocytosis. (Reproduced with permission from Barcellini W, Fattizzo B. Clinical Applications of Hemolytic Markers in the Differential Diagnosis and Management of Hemolytic Anemia. Dis Markers. 2015;2015:635670. doi: 10.1155/2015/635670. Epub 2015 Dec 31. PMID: 26819496; PMCID: PMC4706900.)

and **Heinz bodies.** Most accurate test is a G6PD level **a**
**ode.** Patients should avoid triggers.

## nal Hemoglobinuria

ns found on the surface of RBCs protect them from com-
emolysis. **PNH is a deficiency in GPI-anchor molecules**
**D59 attached to cells, resulting in hemolysis by com-**
manifest as anemia, dark urine, venous thrombosis, and
**ost accurate diagnostic test is CD55/CD59 absence via**

## cytosis

nt defect or deficiency in **spectrin or ankyrin,** an RBC
resulting in a loss of RBC membrane surface area →
take spherical shapes and are subsequently destroyed by
ly presents as an extravascular hemolytic anemia. Best ini-
with smear showing **spherocytes** (see Figure 2.7-9). Most
test is an **osmotic fragility test.** Manage with a **splenec-**

**ssive** disorder caused by a mutation of adult hemoglo-
s Glu replaced by Val). Signs and symptoms are due to
d a tendency of sickled cells to aggregate and cause **vaso-**

A classic
deficien
presenti
SOB afte

Causes
deficien
acidosis
dapsone
nitrofura

Patients
for any
lifelong
pneumo
Haemop
operatio

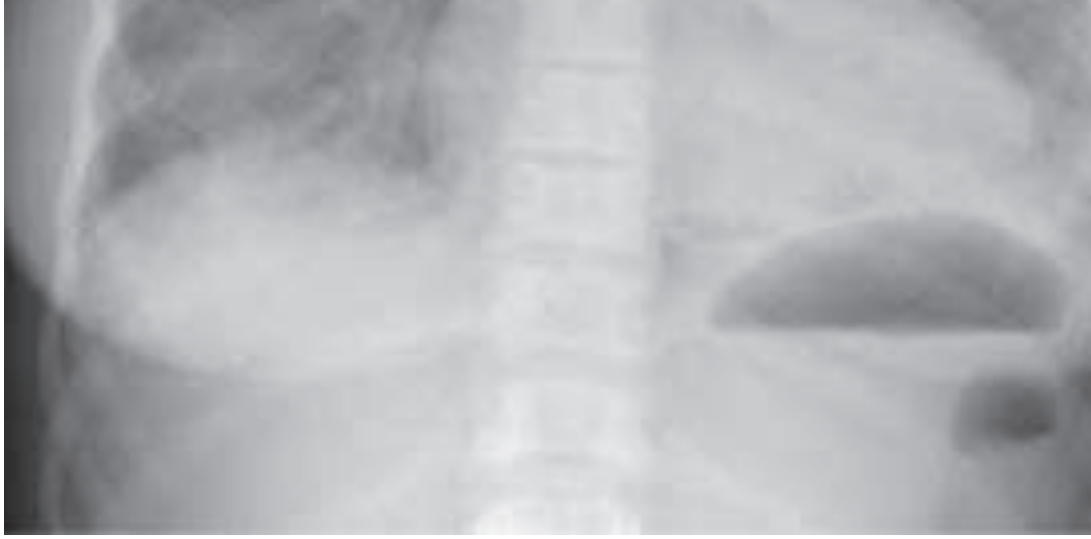


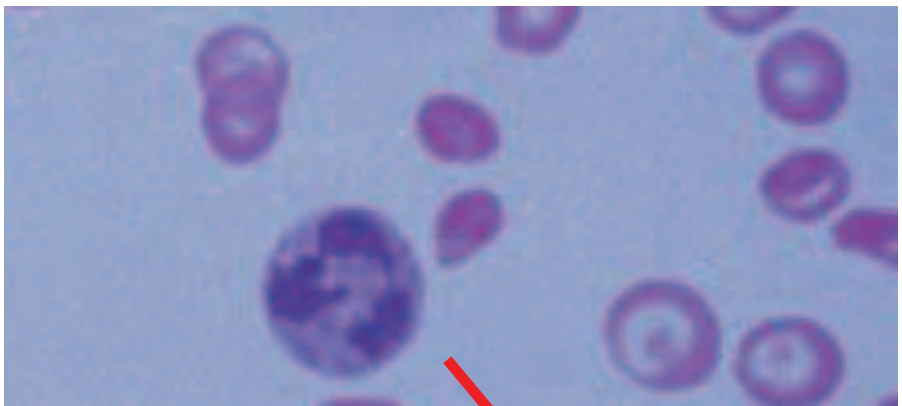
**FIGURE 2.7-10. Acute chest syndrome.** Frontal CXR of a 19-year
cell disease and acute chest pain. Note the bilateral lower and midlung
diomegaly. (Reproduced with permission from USMLE-Rx.com.)

- Other potential complications include splenic sequest
  ing of blood into the spleen resulting in hypovolemia
  (2° to infection with viruses such as parvovirus B19
  tions both present with ↓ hematocrit but are disting
  ↓ platelets in aplastic crisis (2° to bone marrow involv
  to ↑ platelets in splenic sequestration.

CD is
. They
crosis

## Diagnosis

Fastest initial test is CBC with smear **showing sickle ce**
bodies (see Figure 2.7-11A, B). Most accurate test is hem
resis.



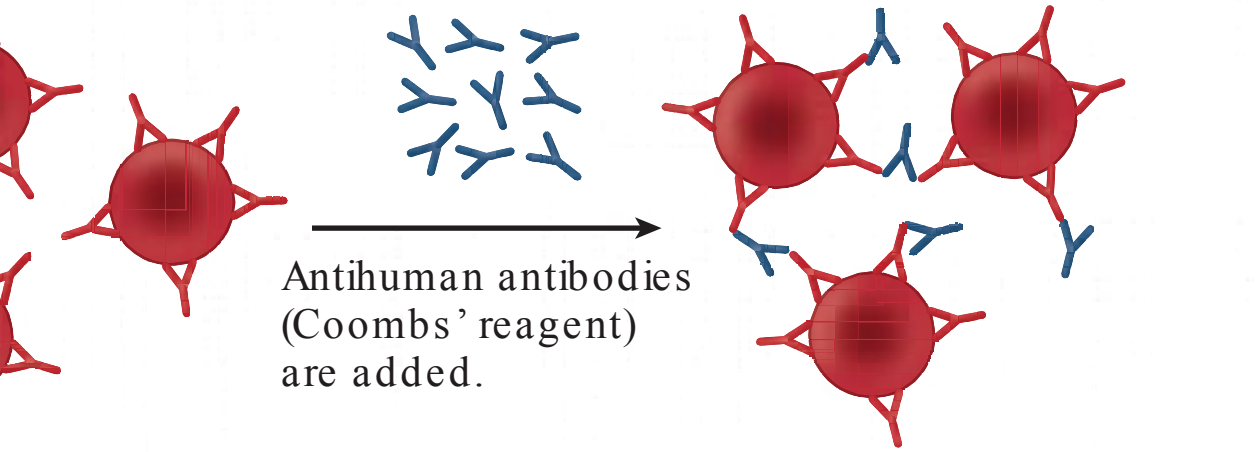


rn of a VOC progressing to acute chest syndrome, initi-
ydration and incentive spirometry, and keep the sickle
his can be done with simple transfusions or, if necessary,
sion in an ICU setting.

## lytic Anemia

nst RBC membrane destroy blood cells, causing hemoly-
n [Ig**G,** associated with SLE, chronic lymphocytic leuke-
and cold (Ig**M,** associated with Mycoplasma pneumonia,
esents as a hemolytic anemia. Diagnose with a **direct**
igure 2.7-12). If AIHA is mild, no treatment is necessary.
is treated with **steroids.** Severe cold AIHA is managed by
cold ± rituximab (anti-CD20 antibody).

ll production due to **destruction of bone marrow cells.**
y, as in **Fanconi anemia;** may have an **autoimmune** or




t's RBCs are
d to remove any
nd antibodies.

Positive test result when RBCs
agglutinate, demonstrating the
presence of bound antibodies
to RBC antigens.

The indi
antibod
serum. T
sensitize

Both AIH
can pres
positive
AIHA wi
**test.**

Diagnosed by clinical presentation and CBC. Most acc
**marrow biopsy** revealing hypocellularity and space occup

### Treatment

**Blood transfusion and stem cell transplantation** to rep
severe cases or in patients without a stem cell donor, im
cyclosporin A and antithymocyte globulin to prevent au
destruction. **Infections** should be treated aggressively.

## THALASSEMIAS

**Hereditary** disorders involving ↓ or absent production
chains of hemoglobin. α-Thalassemia is caused by a muta
of the four genes that encode α-hemoglobin; β-thalasse
mutation of one or both of the two genes encoding β-hem

### History/Pe

Thalassemia is most common among people of **African, M**
**Asian descent.** Disease presentation varies with the numb
(see Table 2.7-4). A typical case can be an asymptomatic
ual with a microcytic anemia and **normal iron studies.**

### Diagnosis

Diagnosed by **hemoglobin electrophoresis** (normal in c
rier) and DNA studies.

### Treatment

- Most patients do not require treatment.
- Those with β-thalassemia major and hemoglobin H dis
  fusion dependent and require iron chelators (defero
  overload.

| | |
|---|---|
| | microcytic and hypochromic on peripheral smear. Common in the Mediterranean. |
| 0/4 α | Patients die in utero. |
| 1/4 α | Patients have severe hypochromic, microcytic anemia with chronic hemolysis, splenomegaly, jaundice, and cholelithiasis. **The reticulocyte count ↑ to compensate,** and one-third of patients have skeletal changes due to expanded erythropoiesis. |
| 2/4 α | Patients have low MCV but are usually asymptomatic. |
| 3/4 α | Patients have no signs or symptoms of disease. |

likely d

ocytosis is associated with hypoxia (lung disease, smok-
es, or a poor intrauterine environment), neoplasia (EPO-
rs), or **polycythemia vera (PCV).**
from **clonal proliferation of a pluripotent marrow stem**
a JAK2 **mutation.** All marrow cell lines ↑ but RBCs are
antly affected.
sorder most commonly affects older individuals.
cytosis is associated with hypovolemia: diuresis, gastroen-

l test is a CBC showing **elevated RBCs/WBCs/platelets**
d EPO level. ↓ **EPO,** normal $O_2$, and **HCT > 60%** sug-

st is the JAK2 **mutation.**

Premed
and dipl
used to
reaction

Hemogl
transfus
tubular
failure

porphyria, erythropoietic porphyria, and porphyria cutan
mon). Lead poisoning inhibits heme synthesis enzymes
**porphyria-like** disorder.

### History/Pe

- In general, porphyrias are characterized by a combinat
  **titis, neuropsychiatric complaints,** and **visceral com**
  form of **colicky abdominal pain and seizures.**
- Physical examination reveals **tachycardia, skin eryth**
  **flexia,** and a nonspecific abdominal examination.
- Patients with the erythropoietic form present with
  Acute attacks are associated with stimulants of ↑ hem
  fasting or chemical exposures; well-known triggers a
  rates, and OCPs. Urine may appear pink or brown afte
  may have a ⊕ family history.

### Diagnosis

- Best initial test is urine and plasma porphyrin levels.
- Most accurate test is specific mutation or enzyme analy

### Treatment

- Avoidance of triggers and symptomatic treatment durin
- High doses of **glucose** ↓ heme synthesis during attack
  back to the heme synthetic pathway).

**eactions**

| onhemolytic reaction | allergic reaction | hemolytic t |
|---|---|---|
| **rmation** during storage | Antibody formation **against donor** | Preformed (acu |

cells (blasts) resulting in unregulated growth and differentia-
e marrow. As the bone marrow becomes overwhelmed, ane-
ion (↓ mature WBCs), and hemorrhage (↓ platelets) result.

lymphocytic leukemia (ALL) affect children as well as
**e most common childhood malignancy.**
progression. Patients present with signs and symptoms of
fatigue), thrombocytopenia **(petechiae, purpura, bleed-**
(ineffective and immature WBCs) and **DIC** (seen in
y expansion into the periosteum may lead to **bone pain**
L).
**hepatosplenomegaly** and **swollen/bleeding gums** from
tion and ↓ platelets. Leukemic cells also infiltrate the skin

CBC with smear showing blast cells.
st is a bone marrow biopsy with flow cytometry to classify

nfiltrated with blast cells is consistent with leukemia. **In**
**emic cells are myeloblasts; in ALL they are lympho-**
lls can be distinguished by morphology (see Figure 2.7-
, and immunophenotyping (see Table 2.7-6).
t can be elevated, but the cells are dysfunctional, and
neutropenic with a **history of frequent infection.** If the
AML is very high (> 100,000/mm$^3$), there is a risk of **leu-**
occluding the microcirculation, leading to pulmonary
nptoms, ischemic injury, and DIC).

and AML are treated with **chemotherapy.** Patients with a



**FIGURE**
The red
in the cy
pathogn
McPhedran

Eighty-f
ALL ach
chemot

A 35-y
ED afte
require
stabiliz
follow
his ho
compl
prolon



B

**Lon peripheral smear. (A) AML.** Large, uniform myeloblasts with round or kidney-s
**(B) ALL.** Peripheral blood smear reveals numerous large, uniform lymphoblasts, wh
lo. Some lymphoblasts have visible clefts in their nuclei. (Used with permission of Dr. Peter M

## Chronic Lymphocytic Leukemia

A malignant, clonal proliferation of **functionally incom**
that accumulate in the bone marrow, peripheral blood, ly
and liver. The most common type of leukemia. Almost al
**differentiated B lymphocytes.** Primarily affects **older adu**
the male-to-female ratio is 2:1.

### History/Pe

Often asymptomatic; patients present with **fatigue, mal**
Common physical findings are **lymphadenopathy and sp**

### Diagnosis

- Best initial test: CBC with differential and smear sh
  **sis** (NK cells, T cells, or B cells > 5000/mm$^3$) and **ch**
  **cells** (fragile leukemia cells crushed by the slide). See
- Most accurate test: Flow cytometry showing the CD
  (normally found on T cells).

A

toms
ltiple
cts

se

**TABLE 2.7-6. Myeloblasts vs Lymphoblasts**

| Variable | Myeloblast |
|---|---|

**thrombocytopenia (worst prognosis).**

s not curable, long disease-free intervals may be achieved reatment of symptoms. The clinical stage correlates with l.

## us Leukemia

of myeloid progenitor cells, leading to leukocytosis with and basophils and sometimes ↑ erythrocytes and platelets CML, the **BCR-ABL** translocation must be present. In his is reflected by the **Philadelphia chromosome t(9,22).** cts **middle-aged** patients.

re asymptomatic at diagnosis. Typical signs and symptoms **mia.**

e **splenomegaly** with LUQ pain and early satiety. **Consti-ms** of weight loss, anorexia, fever, and chills may also be

ML go through three disease phases:

ut treatment this phase typically lasts 3.5–5.0 years. Infec-g complications are rare.

ransition toward blast crisis, with an ↑ in peripheral and ood counts. Should be suspected when the differential t ↑ in basophils and thrombocytopenia (platelet count

rge percentage of untreated CML patients will eventually Resembles acute leukemia; survival is 3–6 months.

est: Philadelphia chromosome via PCR or FISH analysis 22) translocation, although some cases lack the transloca-

s a **very high WBC count**—often > 100,000/mm³ at diag-

**teristic s** small, m cells (ar cytes are preparat permission of Hemato

Look for toward the cove leukemi

Likely di presenta

- **ALL** any
- **AM** in ar
- **CM**
- **CLL**

A comm **lympho**

**older men.**

### History/Pe

- Typically presents with pancytopenia, bone marrow i
  nomegaly.
- Patients complain of weakness, fatigue, petechiae,
  (especially with atypical mycobacteria such as Myc
  intracellulare), abdominal pain, early satiety, and wei
  are similar to those of CLL except that patients rarely h
  thy.

### Diagnosis

- Best initial test: CBC with smear showing pathognor
  (mononuclear cells with many cytoplasmic projection
  that stain with **tartrate-resistant acid phosphatase (T**
  can sometimes be seen as well.
- Most accurate test: Flow cytometry identifying the "hai

### Treatment

- First-line therapy is **cladribine.** If left untreated, most p
  progressive pancytopenia and splenomegaly eventually
- Alternative treatment options include splenectomy and
- The median survival without treatment is 5 years.

## LYMPHOMAS

Malignant transformations of lymphoid cells residing pri
tissues, especially the lymph nodes. Classically organized
and non-Hodgkin (NHL) lymphoma (see Table 2.7-7).

### Non-Hodgkin Lymphoma (NHL)

| | |
|---|---|
| association. | EBV association. |

n from Le T et al. First Aid for the USMLE Step 1 2015. New York, NY:

**Hodgkin Lymphoma Types**

| | |
|---|---|
| **B-cell neoplasms** | |
| Adults (mean age 55) | ▪ **Indolent course or low grade.**<br>▪ Painless waxing and waning adenopathy<br>▪ Localized disease (15%) may be cured wi |
| Usually middle-aged and elderly | ▪ **Intermediate grade.**<br>▪ **Most common NHL in adults.**<br>▪ Often presents with single rapidly growi<br>▪ High cure rate with **R-CHOP therapy.** |
| Children and adolescents | ▪ **High grade,** "starry sky" appearance on l<br>▪ Jaw lesion in Africa, abdominal lesion in<br>▪ Associated with **EBV** and t(8;14) transloc<br>▪ Aggressive treatment with chemotherap |
| Elderly men | ▪ CD5+.<br>▪ Rarest form of NHL. |
| **T-cell neoplasms** | |
| Adults | ▪ **High grade,** can progress to ALL.<br>▪ Presents with cutaneous lesions. |

- Treatment is based on histopathologic classification and consists of radiation, chemotherapy, or both.
- Generally, for low-grade indolent NHL, treatment is pa
- For high-grade aggressive NHL, treatment is aggress approach.

## Hodgkin Lymphoma

A **predominantly B-cell malignancy** associated with EBV **age distribution:** age 30 (primarily the nodular sclerosin (mainly the lymphocyte-depleted type). It has a male prec hood.

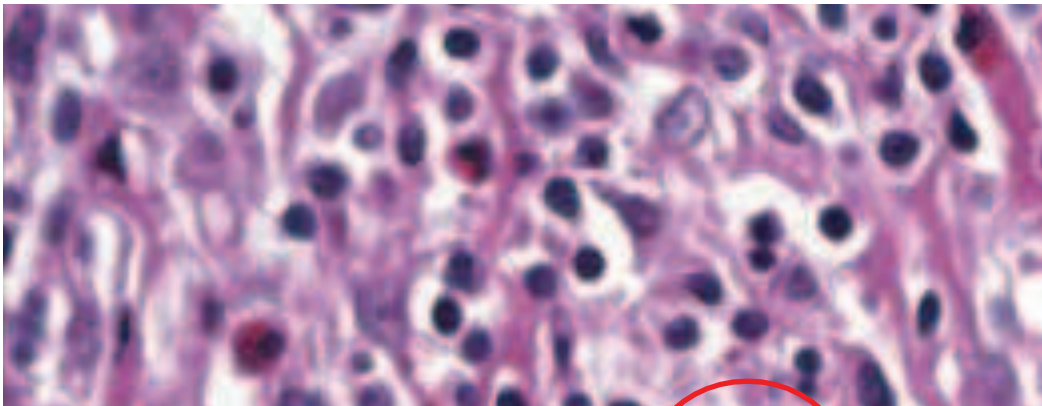
### History/Pe

- HL commonly presents **above the diaphragm (classic nopathy;** see Figure 2.7-17A). Infradiaphragmatic in disseminated disease.
- Patients can have **systemic B symptoms** (see Table 2 **hepatosplenomegaly. Pel-Ebstein fevers** (1–2 weeks nating with 1–2 afebrile weeks) and **alcohol-induced** are rare signs specific for HL.

### Diagnosis

- Best initial step is an **excisional lymph node biops** sic **Reed-Sternberg cells** (giant abnormal B cells with





# ell Disorders

## IA

of malignant plasma cells with **excessive production of noglobulins** (typically **ineffective** IgA or **IgG**) or **immu-nts** (kappa/lambda light chains). MM primarily affects the the seventh decade. Risk factors include radiation, mono- of undetermined significance (MGUS), and, possibly, and pesticides.

of MM patients present with **bone pain** or with a patho- MM cells infiltrate bone marrow, where they **activate** ting **lytic lesions** and weak bones). **Anemia, hypercalce- bnormalities** are also seen.
ne to **infection** (IgG and IgA produced by myeloma cells thus making them **ineffective**) and have **elevated mono- eins** in the serum and/or urine.

**Serum protein electrophoresis** showing **IgG** or **IgA** es (see Figure 2.7-18).
est: Bone marrow biopsy **showing > 10% monoclonal** cells.
may show **rouleaux** formation, whereas a urinalysis may **es protein** (paraprotein). **Total protein:albumin gap is**

is insufficient for the diagnosis of MM, as MGUS, CLL, denström macroglobulinemia, and amyloidosis can also

For MM

hyperCa
Renal inv
Anemia
Bone lyti

MGUS is
plasma c
may eve
(1–2% p

Hyperca
bone an
polyuria

FIGURE
Serum p
showing
tic of M
IgM ind
linemia.



A B

**FIGURE 2.7-19. Multiple myeloma skeletal survey.** Characteristi
multiple myeloma involving the tibia and fibula **(A)** and the skull **(B)** a
with permission from Lichtman MA et al. Williams Hematology, 8th ed. New York, NY: McGraw
with permission from Kantarjian HM, Wolff RA, Koller CA. MD Anderson Manual of Medical Onc
McGraw-Hill; 2011.)

## WALDENSTRÖM MACROGLOBULINEMIA

A clonal disorder of B cells that leads to a malignant mo
thy. ↑ **levels of IgM** result in **hyperviscosity syndrome,**
malities, cryoglobulinemia, cold agglutinin disease (lead
hemolytic anemia), and amyloidosis. Tissue is infiltrated b
tic plasma cells. A **chronic, indolent disease of the elderl**

### History/Pe

- Presents with nonspecific symptoms: lethargy, weight
  **phenomenon** from cryoglobulinemia. **Neurologic**
  from mental status changes to sensorimotor periphe
  blurry vision (**engorged blood vessels** can be noted on
  seen. Organomegaly and organ dysfunction can be pre
- As with multiple myeloma, **MGUS is a precursor** to d

### Diagnosis

- Most accurate test: **Bone marrow biopsy and aspir**
  abnormal plasma cells, classically with **Dutcher b**
  deposits around the nucleus). Serum and urine pro

| |
|---|
| eposition of $\beta_2$-microglobulin, which accumulates in patients on long-rm dialysis. |
| eposition of abnormal gene products (eg, transthyretin, aka ealbumin). A heterogeneous group of disorders. |
| eposition of otherwise normal transthyretin. |

= 1° amyloidosis.

ation depends on the type, amount, and tissue distribution ne most common forms of systemic amyloidosis, 1° (AL) major sites of clinically important amyloid deposition are **eart, and liver.**

s, amyloid deposition is limited to one organ (the brain in **se**).

Tissue biopsy with **Congo red staining** showing apple- under polarized light. Immunohistochemistry can iden-tein type.

reated with experimental chemotherapy and autologous ; in 2° amyloidosis, the underlying condition should be

Q

A 71-y
marke
that ar
dull ba
times,
have b
of an 1
likely r

- Follow neutropenia clinically with a CBC and ANC
  nia or anemia is present, bone marrow biopsy and asp
  formed.
- **Serum immunologic evaluation, ANA levels, and a v**
  **vascular disease may be merited.**

### Treatment

- **Infection management:** Neutropenic patients can't
  inflammatory response.
- Fever in the context of neutropenia (neutropenic
  **emergency** and should be treated immediately with **b**
  **biotics such as cefepime.** Suspected fungal infection
  appropriately as well.
- Hematopoietic stem cell factors such as **G-CSF** can
  the duration of neutropenia. Rarely, **IVIG and alloge**
  **transplantation** may be used.

## Eosinophilia

An **absolute eosinophil count > 350/mm.** Eosinophilia
the overproduction of cytokines (IL-3, IL-5, and GM-CSF
that stimulate the migration of eosinophils into peripher
Eosinophilia may be a 1° disorder but is usually 2° to
most common cause in the developed world is **allergy,** w
oping world it is **parasitic infection.**

### History/Pe

- A **travel, medication, atopic, lymphoma/leukemia**
  should be elicited.
- Exam varies by cause. Patients with hypereosinophil

Transplantation from a donor to a genetically different

ransplantation between identical twins (from a donor to a entical patient).

donation, efforts are made to ABO and HLA match the ent. Despite matching and immunosuppression, however, be rejected. There are three types of solid organ rejection: e, and chronic (see Table 2.7-10).

ease (GVHD) is a complication specific to allogeneic nsplantation in which donated T cells attack host tissues, in, liver, and GI tract. It may be acute (< 100 days post- ronic (> 100 days afterward).

**compatibility antigens are thought to be responsible for** ch presents with **skin changes, cholestatic liver dysfunc- tive lung disease,** or **GI problems.**

reated with high-dose corticosteroids.

splant immunosuppression regimens include: prednisone, mofetil (MMF), FK506 (tacrolimus) to suppress immune- on, TMP-SMX, ganciclovir, and ketoconazole to prevent tion in the immunosuppressed host.

VHD is the **graft-vs-leukemia effect,** in which leukemia treated with an allogeneic bone marrow transplant have

**es of Solid Organ Transplant Rejection**

| | **hyperacute** | **acute** | |
|---|---|---|---|
| t | Within minutes. | Between 5 days and 3 months. | Mo |
| | Preformed antibodies. | T-cell mediated. | Chr fib |
| | | | |

**sociated With Neoplasms**

| ndition | neoplaSM |
|---|---|
| | Squamous cell and basal cell carcinomas of the |
| | Squamous cell carcinoma of the skin. |
| | Malignant melanoma. |
| | ALL ("We will **ALL** go **Down** together"). |
| | Malignant lymphomas. |
| | Aggressive malignant NHLs and Kaposi sarcom |
| sthenia gravis) | Benign and malignant thymomas. |
| icious anemia, postsurgical gastric | Gastric adenocarcinoma. |
| eflux | Esophageal adenocarcinoma. |
| ophic glossitis, esophageal webs, y) | Squamous cell carcinoma of the esophagus. |
| | Colonic adenocarcinoma. |
| Wilson disease, etc) | Hepatocellular carcinoma. |
| mentation and epidermal | Visceral malignancy (stomach, lung, breast, ute |

196 Syphilis
196 Genital Lesions
199
200 Genitourinary Infections
201 Urinary Tract Infections
202 Pyelonephritis
203 Hematologic Infections
Sepsis
204 Malaria
204 Infectious Mononucleosis
206
207 Fever
Fever of Unknown Origin
ency Virus 208 Neutropenic Fever
ns 210 Tick-Borne Infections
asis (Thrush) 211 Lyme disease
s 211 Rocky Mountain Spotted Fever
211
eumonia 212 Infectious conjunctivitis
213 orbital cellulitis
mplex 214 otitis Externa
214
Diseases 215 Infective Endocarditis
215 Anthrax
216 osteomyelitis

yellow-green sputum or hemoptysis), dyspnea, nig
**chest pain.**
- **Atypical symptoms:** Gradual onset, dry cough, h
sore throat, GI symptoms.

- Lung examination may show ↓ or bronchial breath so
ing, dullness to percussion, egophony, and/or tactile fre
- Elderly patients and those with COPD, diabetes, or im
may have minimal or atypical signs on physical examin

### Diagnosis

- Diagnosis requires two or more symptoms of acute r
plus a new infiltrate on CXR or CT.
- Workup includes physical examination, **CXR** (see F
Most cases are treated empirically in the outpatient set
stain and culture (see Figure 2.8-2), nasopharyngeal
ture, and ABG are obtained in minority of cases, mo
patients or outpatients with persistent symptoms.
- Tests for specific pathogens include the following:
  - Legionella: Urine Legionella antigen test, sputum
fluorescent antibody (DFA), culture.
  - Chlamydophila (Chlamydia): Serologic testing,
chain reaction).
  - Mycoplasma: Usually clinical. Serum cold aggl
Mycoplasma antigen may also be used.
  - S pneumoniae: Urine pneumococcal antigen test, c
  - **Viral:** Nasopharyngeal aspirate, rapid molecular tes
influenza, respiratory syncytial virus), DFA, viral cu

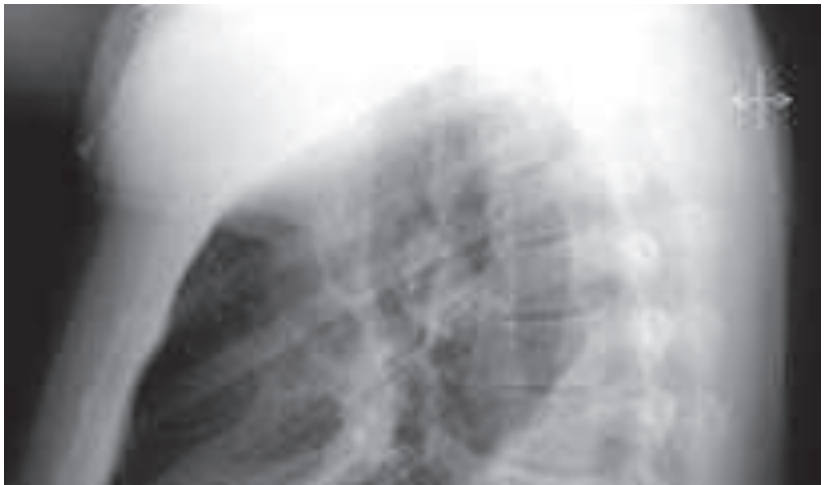
**ses of Pneumonia by Age**

| Children | Adults | Adults |
|---|---|---|
| (Weeks–18 years) | (18–40 years) | (40–65 years) |

| | |
|---|---|
| | Anaerobes. |
| rs | Spneumoniae, Klebsiella, Staphylococcus. |
| | Staphylococcus, Pseudomonas, Burkholderia, mycobacteria. |
| | Hinfluenza , Moraxella catarrhalis, Spneumoniae. |
| | Staphylococcus, Spneumoniae, Hinfluenza . |
| | GBS, Ecoli, Listeria. |
| | Obstruction, bronchogenic carcinoma, lymphoma, Wegener granulomatosis, immunodeficien y, unusual organisms (eg, Nocardia, Coxiella burnetii, Aspergillus, Pseudomonas). |

izes the recommended initial treatment for pneumonia. ment with oral antibiotics is recommended in uncompli-





The pne
given to
old, peo
(eg, sick
immunc
patients

A B

**FIGURE 2.8-2. Common pathogens causing pneumonia. (A)** S a
of gram-⊕ cocci were isolated from the sputum of a patient who develo
hospitalized. **(B)** S pneumoniae. Sputum sample from a patient with p
characteristic lancet-shaped gram-⊕ diplococci.

- For patients with obstructive diseases (eg, CF or bron
adding pseudomonal, staphylococcal, or anaerobic cov

### Complications

Pleural effusion, empyema, lung abscess, necrotizing pneu

**Pneumonia**

| | Suspected pathogens | Empiric |
|---|---|---|
| d<br>ise healthy,<br>ths. | Spneumoniae, Mycoplasma pneumoniae, Cpneumoniae, Hinfluenza , viral. | Macrolide or doxycyc |
| D, heart<br>ver disease,<br>se within | Spneumoniae, Hinfluenza , aerobic GNRs (Ecoli, Enterobacter, Klebsiella), Saureus, Legionella, viruses. | Fluoroquinolone or β- |
| nia requiring | Spneumoniae, Hinfluenza , anaerobes, aerobic GNRs, Legionella, Chlamydia. | Fluoroquinolone or a<br>β-lactam + macrolide |
| nia requiring | Spneumoniae, Legionella, Hinfluenza , anaerobes, aerobic GNRs, Mycoplasma, | Antipneumococcal β-<br>azithromycin or fluo |

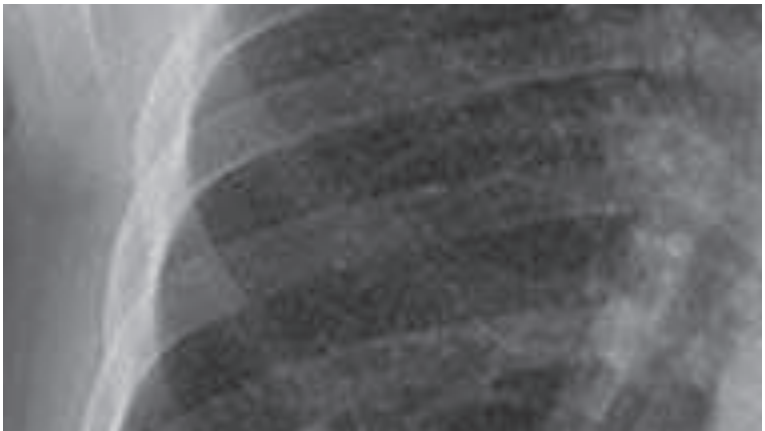
coholism, preexisting lung disease, diabetes, advancing

**TB exposure in the United States: Homelessness** and onditions (eg, **prisons**), emigration/travel from developing ment in a health profession, interaction with known TB

ough, **hemoptysis,** dyspnea, **weight loss, fatigue, night** chexia, hypoxia, tachycardia, lymphadenopathy, an abnor- ation, and prolonged (> 3 weeks) symptom duration.
cause of fever of unknown origin (FUO).
n present with atypical signs and symptoms and have xtrapulmonary TB.

Mycobacterial culture of sputum (or blood/tissue for extra- se) is the gold standard but can take weeks to obtain. A **t stain** (see Figure 2.8-3) can yield rapid preliminary sensitivity.
mmon finding among typical hosts is a cavitary infiltrate lobe on CXR (see Figure 2.8-4), which may be accom- ication of one or more nearby lymph nodes **(Ghon com-**





The inte
of the P
patients
vaccine



**FIGURE**
the red c
bacilli o
with permi
Histopatho
Lange; 199

### Treatment

All cases (both latent and active) must be reported to lo
departments. Respiratory isolation should be instituted
pected. Treatment measures are as follows:

- **Active disease:**
  - Directly observed multidrug therapy with a four-d
    **pyrazinamide, rifampin, ethambutol)** × 2 month
    and rifampin × 4 months.
  - Administer **vitamin $B_6$** (pyridoxine) with **INH** to
    neuropathy.
- **Latent disease:** For a ⊕ PPD without signs or symp
  ease, treat with **INH × 9 months.** Alternative regimen
  months or rifampin × 4 months.

## ACUTE PHARYNGITIS

Viral causes are more common (90% in adults), but it is i
streptococcal pharyngitis **(group A β-hemolytic** Streptoco
ologies are as follows:

- **Bacterial:** Group A streptococci (GAS), Neisseria gonc
  terium diphtheriae, M pneumoniae.
- **Viral:** Rhinovirus, coronavirus, adenovirus, HSV, EB
  virus, coxsackievirus, acute HIV infection.

dermally on the volar surface of the forearm. The diameter of indura
rs. BCG vaccination typically renders a patient PPD ⊕ but should
mmended for unvaccinated individuals. The size of induration tha
as follows:

of four Centor criteria are met (see Table 2.8-4), the sen-
gen testing is > 90%.

l, begin empiric antibiotic therapy with **penicillin** × 10
ns, amoxicillin, and azithromycin are alternative options.
be attained with fluids, rest, antipyretics, and saltwater

**:** Acute rheumatic fever (see the Cardiovascular chapter),
l glomerulonephritis.
ervical lymphadenitis, mastoiditis, sinusitis, otitis media,
or peritonsillar abscess, and, rarely, thrombophlebitis
ein **(Lemierre syndrome)** due to Fusobacterium, an oral

**scess** may present with odynophagia, trismus ("lockjaw"),
potato" voice, unilateral tonsillar enlargement, and ery-
uvula and soft palate deviated away from the affected side.
fluid and localize the abscess via intraoral ultrasound or
ntibiotics and **surgical drainage.**

tion of the paranasal sinuses. The maxillary sinuses are
ected. Subtypes include the following:

**fied entor Criteria (Centor Criteria + Age)**

| **iteria** | **points** |
|---|---|
| | 1 |

**thema.**
most co
yngitis.
Disease Co

tening
r
oorly
mmune



ronal
maxil-
bony
ic max-
ission from
Otolaryn-
w York, NY:

over the affected area.
- High fever, leukocytosis, and a purulent nasal dischar acute bacterial sinusitis.

### Diagnosis

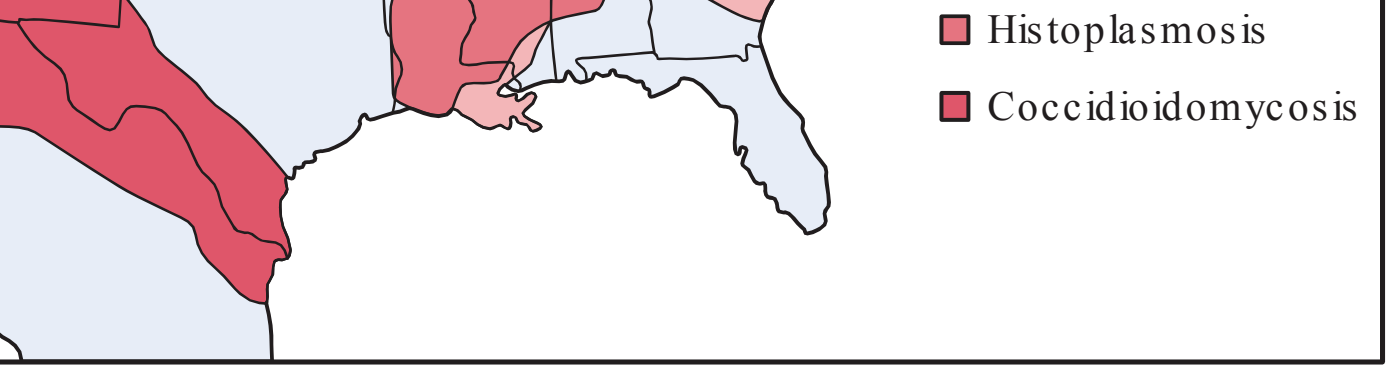
- A clinical diagnosis. Culture and radiography are ger for acute sinusitis but may guide the management of cl
- Transillumination shows opacification of the sinuses (l
- CT is the test of choice for sinus imaging (see Figure necessary only if symptoms persist after treatment.

### Treatment

- Most cases of acute sinusitis are viral and/or self-limi with symptomatic therapy (decongestants, antihistar lavage, pain relief).
- **Acute bacterial sinusitis:** Consider amoxicillin/clavu or clarithromycin, azithromycin, trimethoprim-sulfar SMX), a fluoroquinolone, or a second-generation ce days.
- **Chronic sinusitis:**
  - Antibiotics are similar to those used for acute dise **ger course** (3–6 weeks) may be necessary.
  - **Adjuvant** therapy with intranasal corticosteroids, antihistamines may be useful in combating the al component of the disease. **Surgical** intervention ma

## COCCIDIOIDOMYCOSIS

A pulmonary fungal infection endemic to the **southwes** (see Figure 2.8-8). Can present as an acute or subacute flulike illness, and may involve extrapulmonary sites, inc




**graphic distribution of systemic fungal infection in the United**
mission from Ryan KJ, Ray CG. Sherris Medical Microbiology, 5th ed. New York, NY: McGraw-

lveolar lavage as well as fungal cultures of sputum, wound affected tissue. Cultures are usually only obtained in hos- or patients with severe disease, and growth can take days to

oides immitis spherules on H&E or other special sputum

ay be normal or may show infiltrates, nodules, cavities, lar adenopathy, or pleural effusion.
oscopy, fine-needle biopsy, open lung biopsy, or pleural is indeterminate.

nazole or itraconazole can be used for mild infection or rapy once the patient is stable. IV therapy is rarely neces- nsider **IV amphotericin B** for severe or protracted 1° pul- and disseminated disease.
eatment is needed for asymptomatic chronic pulmonary ies. Progressive cavitary or symptomatic disease usually plus long-term azole therapy for 8–12 months.

Conside
patient
States w
infection
pregnan
of disse

### Diagnosis

**Best initial step in management:** Rapid influenza test o
nasopharyngeal swab. More definitive diagnosis can be m
viral culture, or PCR assays. Rapid influenza tests have
influenza is usually a clinical diagnosis. Leukopenia is a c

### Treatment

- Symptomatic care with analgesics and cough medicine
- Antivirals such as oseltamivir or zanamivir are most
  **within 2 days of onset** and may shorten the duration
  days.

### Complications

Severe 1° viral pneumonia, 2° bacterial pneumonia (see
2.8-2), sinusitis, bronchitis, and exacerbation of COPD and

## CNS Infections

### MENINGITIS

Acute bacterial meningitis is a **life-threatening** emergenc
"aseptic") meningitis is more common and clinically less
for meningitis include recent ear infection, sinusitis, i
recent neurosurgical procedures, crowded living condition
military), and sick contacts. Causes by age group are listed

**TABLE 2.8-5. Causes of Meningitis by Age Group[a,b]**

eurologic deficits).
HSV); **cryptococcal antigen** (for HIV patients).
ndicated in a minority of patients before LP, in particular
d mental status, papilledema, or focal neurologic deficits
ss lesion or increased intracranial pressure. If CT is being
c antibiotics should be started beforehand. Obtain blood
ay reveal leukocytosis; CSF findings vary (see Table 2.8-6).

Acute b
threaten
should b

ld be administered rapidly (see Table 2.8-7) and can be
y up to 2 hours before an LP.
iral meningitis can be treated with supportive care and

**rofile**

| **rbcs** (per MM$^3$) | **Wbcs** (per MM$^3$) | **glucose** (Mg/dl) | **protein** (Mg/dl) | **opening pressure** (cM $h_2o$) |
|---|---|---|---|---|
| < 10 | < 5 | − 2/3 of serum | 15–45 | 10–20 |
| ↔ | ↑ (> 1000 PMNs) | ↓ | ↑ | ↑ |
| ↔ | ↑ (monos/ lymphs) | ↔ | ↔ or ↑ | ↔ or ↑ |
| ↔ | ↑ | ↔ | ↔ or ↑ | ↔ |
| ↑↑ | ↑ | ↔ | ↑ | ↔ or ↑ |

| | | |
|---|---|---|
| | | or cefo |
| > 60 years/alcoholism/ chronic illness | Pneumococci, gram-⊖ bacilli, Listeria, meningococci. | Ampici cefotax |

- Close contacts of patients with meningococcal menin **rifampin, ciprofloxacin,** or ceftriaxone.
- **Dexamethasone** ↓ mortality, hearing loss, and short-te plications in bacterial meningitis caused by S pneumo minutes before antibiotics.
- If immunocompromised, elderly, or neonate, add amp

### Complications

**Sensorineural hearing loss,** mental impairment, seizur and ↑ intracranial pressure (ICP), subdural empyema, brain abscess, ventriculitis/hydrocephalus, focal neurolog nial nerve palsies), hyponatremia, coma, death.

## ENCEPHALITIS

**HSV** and **arboviruses** are the most common causes of en ologies include CMV, toxoplasmosis, West Nile virus, VZ sia, Legionella, enterovirus, Mycoplasma, and cerebral ma the elderly are the most vulnerable.

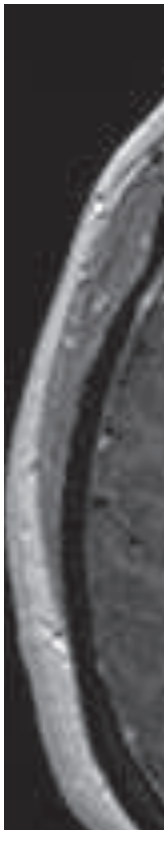
### History/Pe

- Presents with **altered consciousness, headache, fever,** argy, confusion, coma, and focal neurologic deficits (c accentuated DTRs) may also be present.
- The differential includes brain abscess, malignan



**V encephalitis.** Coronal FLAIR image of a young man with HSV haracteristic MRI pattern within the cortex of the right temporal lobe al lobe is also involved (arrow), but to a lesser extent. (Reproduced with per-rison's Principles of Internal Medicine, 17th ed. New York, NY: McGraw-Hill; 2008.)

**is:** Requires immediate IV acyclovir.
**tis:** Treat with IV ganciclovir ± foscarnet.
e for suspected Rocky Mountain spotted fever or ehrlichi- encephalitis with ceftriaxone.



infection of the brain parenchyma, usually with a **"ring-** ance due to fibrous capsule (see Figure 2.8-11). The most are **streptococci, staphylococci,** and **anaerobes; multi-** ften implicated (80–90% are polymicrobial). Nonbacterial plasma and Candida; Aspergillus and zygomycosis should mmunocompromised hosts, and neurocysticercosis should evant epidemiologic settings (Central and South America, and Asia). Modes of transmission include the following:

Due to paranasal sinusitis (10% of cases; frequently affects often due to Streptococcus milleri), **otitis media** or **mas-** **dental infection** (2%).
**on:** Affects patients with a history of head trauma or neu- dures.

**FIGURE**
Post con
ring-enh
frontal l
(smaller
also note
USMLE-Rx.

tastatic
red in

n a
brain
ation.

Lesions < 2 cm can often be treated medically.

- Administer a third-generation cephalosporin + metron cin; give IV therapy for 6–8 weeks. Obtain serial CT/I lution.
- **Dexamethasone** with taper may be used in severe edema; **IV mannitol** may be used to ↓ ICP. Give prop sants.

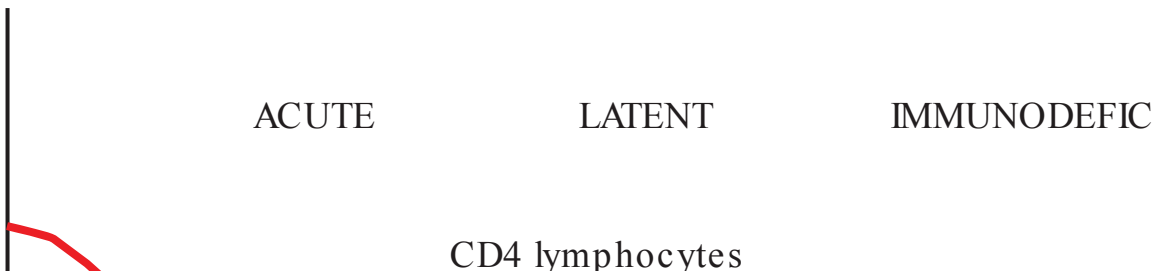
## Human Immunodeficiency Viru

A retrovirus that targets and destroys CD4+ T lymphocyte acterized by a high rate of viral replication that leads to a in CD4+ count (see Figure 2.8-12).

- **CD4+ count:** Indicates the **degree of immunosuppr** apy and prophylaxis, and helps determine prognosis.
- **Viral load:** May predict the **rate** of disease progressi tions for treatment and gauges response to antiretrovira

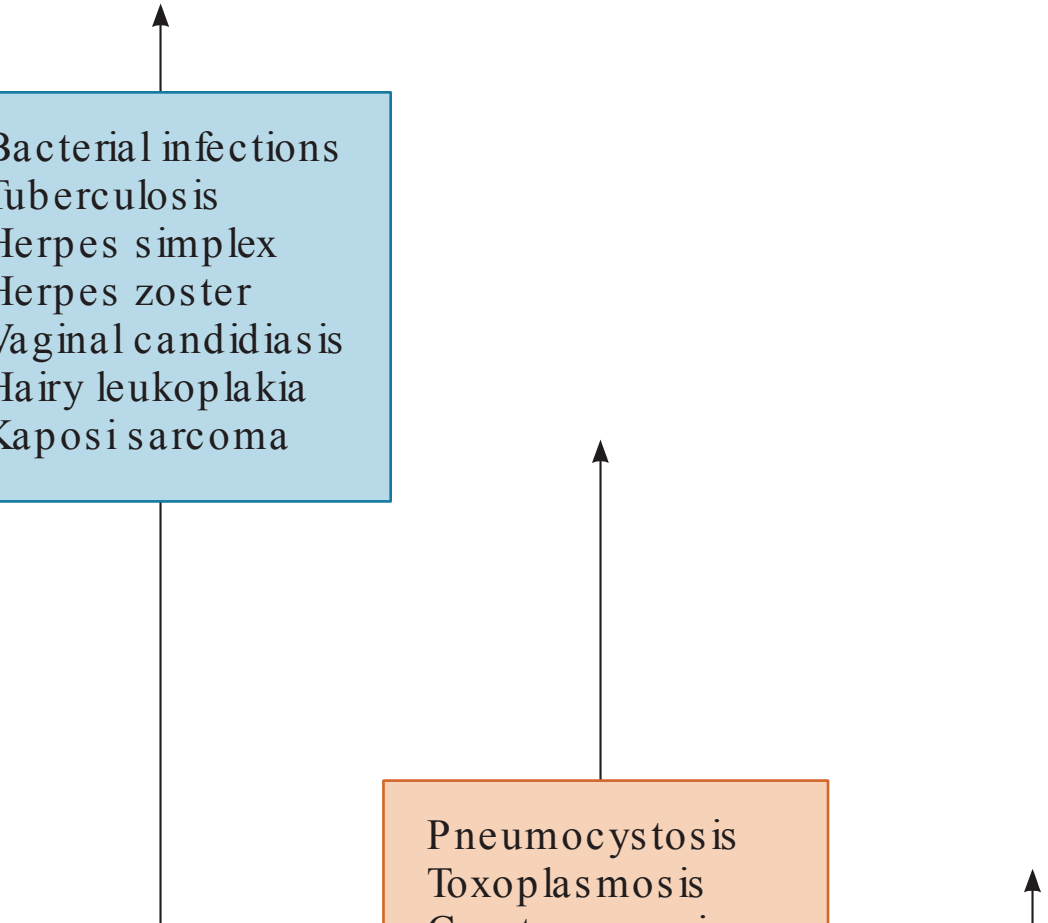
### History/Pe

- Acute HIV infection (acute infection/seroconversion, drome) occurs days to weeks after exposure. The initi **asymptomatic,** but patients may also present with **mo flulike symptoms** (eg, fever, lymphadenopathy, macul yngitis, diarrhea, nausea/vomiting, weight loss, headacl
- HIV infection can later present as night sweats, weig



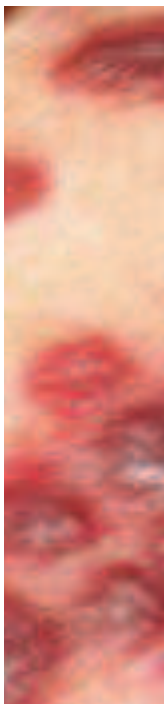
tatus exam, VDRL/RPR, and serologies for CMV, viral
smosis, and VZV.
cute retroviral syndrome (acute HIV) should include **HIV**
load); ELISA may be ⊖.

offered to all HIV-infected patients, regardless of symp-
of AIDS-defining illness or HIV-related conditions) and
dence for decreased morbidity and mortality is most well
atients with CD4 < 500. Patients should be informed of
potential risks of early treatment, importance of adher-
it to lifelong therapy.
men should generally consist of a combination of two
eotide reverse transcriptase inhibitors (RTIs) plus either
ide RTI or one protease inhibitor or one integrase inhibi-
**important principle is to select multiple medications**




**positive**
John Molin
ease Contr

Commo
- Esop
- CM
- Kap
herp
- CNS
- P jir
bact
- HIV
- Diss
fung
- Inva



**FIGUR**

eaction.

with an HIV-⊕ source, begin ART as soon as possib
drug regimen or an expanded regimen of three or mor
depending on the severity of the source infection.

Table 2.8-8 outlines prophylactic measures against
tions.

he only
n to
ot give
ents or

## Opportunistic Infections

Figure 2.8-16 illustrates the microscopic appearance of so
tunistic organisms.

**r HIV-Related Opportunistic Infection**

| dication For prophylaxiS | treatMent | |
|---|---|---|
| 200/mm³, prior P jirovecii<br>n. | Single-strength TMP-SMX. | Discontinue p<br>is > 200/mm³ |
| 50–100/mm³. | Weekly azithromycin. | Discontinue p<br>is > 100/mm³ |
| 100/mm³ + ⊕ IgG<br>ies. | Double-strength TMP-SMX. | — |
| mm or "high risk" (see TB<br>). | INH × 9 months (+ pyridoxine) or<br>rifampin × 4 months. | Include pyrid<br>containing re |

Aspergillus

Cryptococcus

**mmon opportunistic organisms.**

## CANDIDIASIS (THRUSH)

rostomia, antibiotic use, denture use, and immunosup-
V, leukemias, lymphomas, cancer, diabetes, corticosteroid
unosuppressive treatment).

with **soft white plaques that can be rubbed off,** with an
se and possible mucosal burning. The differential diagno-
hairy leukoplakia (affects the lateral borders of the tongue;
d off). Odynophagia is characteristic of candidal esophagi-

nical. KOH or Gram stain shows **budding yeast and/or**

with local therapy (eg, nystatin suspension, clotrimazole
azole such as fluconazole). Treat candidal esophagitis
erapy.

## NINGITIS

lude AIDS and exposure to **pigeon droppings.**

s with headache, fever, impaired mentation, signs of
and **absent meningeal signs.** The differential includes
ymphoma, TB meningitis, AIDS dementia complex,
phalitis, and other fungal disease.

glucose; ↑ protein; ↑ leukocyte count with monocytic
↑ opening pressure); ⊕ **cryptococcal antigen testing in**
**od, CSF India ink stain,** and fungal culture.

AIDS pa

**The Ma**
**Concer**
**Collap**

**T**oxoplas
**M**ycobac
**P**neumoc
**C**andida
**C**ryptoco
**T**ubercu
**C**MV
**C**ryptosp

The CSF
mening
specific.



macro-
st cells
emsa
cClenan,

ram-
hat
CNS
ed
of



Branch-
dapted
rdia
ssisted

Diagnosis

- **CXR** shows diffuse nodular densities, focal infiltra lymphadenopathy (chronic infection is usually cavitary
- The **urine and serum polysaccharide antigen test** i test for making the initial diagnosis of disseminated response to therapy, and diagnosing relapse. Cultur (blood, sputum, bone marrow, CSF).
- The yeast form is seen with special stains on biopsy (b node, liver) or bronchoalveolar lavage (see Figure 2.8-1

Treatment

Depends on the severity of disease and the host:

- **Mild pulmonary disease or stable nodules:** Treat immunocompetent host. Consider itraconazole.
- **Chronic cavitary lesions:** Give itraconazole for > 1 yea
- **Severe acute pulmonary disease or disseminated amphotericin B** or amphotericin B × 14 days followed year or longer. Lifelong maintenance therapy with da be necessary.

## PNEUMOCYSTIS JIROVECII PNEUMONIA

Formerly known as Pneumocystis carinii pneumonia, o include impaired cellular immunity and AIDS.

History/Pe

- Presents with **dyspnea on exertion,** fever, a **nonprodu** pnea, weight loss, fatigue, and **impaired oxygenatio toms have been present for weeks.**
- Can also present as disseminated disease or as local di systems.



B

eumocystis **pneumonia. (A)** Lung tissue stained with silver uncovers omma-shaped spores. **(B)** Frontal CXR shows diffuse "ground-glass" stic of PCP in this patient with AIDS and a CD4+ count of 26. (Part A m Ryan KJ, Ray CG. Sherris Medical Microbiology, 5th ed. New York, NY: McGraw-Hill; 2010. Part om USMLE-Rx.com.)

**dose** TMP-SMX × 21 days.
per should be used in patients with moderate to severe < 70 mm Hg or an arterial-alveolar oxygen gradient > 35).

S

**adults in the United States have been infected with** asymptomatic; reactivation generally occurs in immuno-nts.

ccurs via **sexual contact, vertical transmission, breast y droplets** in nursery or day care facilities, and **blood**

reactivation include the first 100 days status post tissue transplant and HIV/AIDS (CD4+ < 100/mm$^3$ or viral pies).

on may resemble EBV mononucleosis (see the discussion nonucleosis).
tations include the following:
**is:** Associated with retinal detachment ("pizza pie" reti-

Q

A 19-y
broug
room,
roomm
compl
and pa
has a p
CSF ex
level o
of 140
of 140
likely c
condit

several demographic groups. The 1° pulmonary form o
**healthy nonsmokers (Lady Windermere syndrome);** a
affects patients with **preexisting pulmonary disease** such
CF. Disseminated infection occurs in AIDS patients with

### History/Pe

- Disseminated M avium infection in AIDS is associate
  **ness,** and **weight loss in patients who are not on**
  **(HAART) or chemoprophylaxis for MAC.**
- Hepatosplenomegaly and lymphadenopathy are occasi
- Adrenal insufficiency is possible in the setting of adren

### Diagnosis

- Obtain mycobacterial **blood cultures** (⊕ in 2–3 weeks
- Labs show anemia, hypoalbuminemia, and ↑ **serum al**
  **and LDH.**
- Biopsy of bone marrow, intestine, or liver reveals **f**
  with **acid-fast bacilli.** Typical granulomas may be abs
  promised patients.

### Treatment

Treat with **clarithromycin** and consider **HAART if dru**
**tol ± rifabutin is second line.** Continue for > 12 mont
is > 100/mm$^3$ for > 6 months.

### Prevention

Weekly azithromycin for those with a CD4+ < 50/mm
opportunistic infection.

## TOXOPLASMOSIS

h a low-dose regimen until the disease has resolved clini-
aphically.
**ctrim DS)** or pyrimethamine + dapsone can be used for
atients with a CD4+ count < 100/mm$^3$ and a ⊕ toxoplas-

## Transmitted Diseases

**bacterial STD in the United States.** Caused by Chla-
which can infect the genital tract, urethra, anus, and eye.
**unprotected sexual intercourse and new or multiple**
xists with or mimics N gonorrhoeae infection (known as
hritis when gonorrhea is absent). LGV serovars of Chla-
granuloma venereum, an emerging cause of proctocolitis.

n asymptomatic in men and may present with **urethri-**
**ent cervicitis,** or **pelvic inflammatory disease (PID)** in

y reveal cervical/adnexal tenderness in women or penile
sticular tenderness in men.
diagnosis includes gonorrhea, endometriosis, PID, orchi-
UTI.
**ma venereum** presents in its 1° form as a **painless,** tran-
hallow ulcer. In its 2° form, it presents as painful swelling
nodes, and in its 3° form it can present as an "anogenital
pruritus with discharge, rectal strictures, rectovaginal fis-
ntiasis).

**FIGURE**
(Adapted v
ity of mole
toxoplasm
gitis in AID
Indian Acad

Ring-en
with AID
consider
CNS lym

Chlamyd
of nong

Chlamyd
neonata
nongon

female reproductive tract. Infection in men tends to be lim

### History/Pe

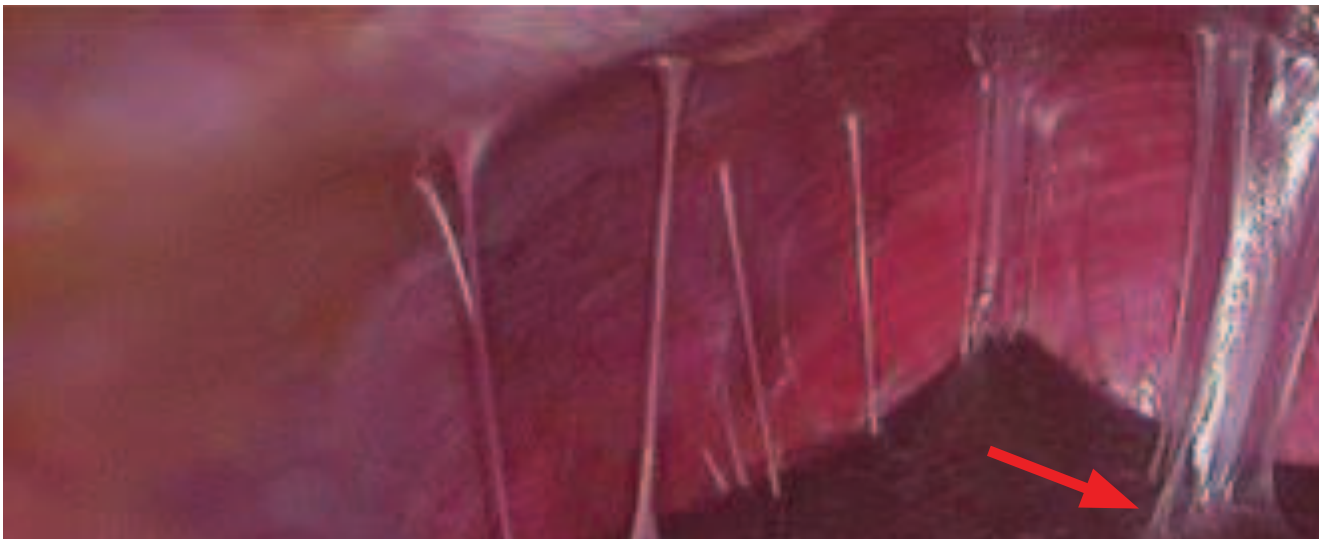
- Presents with a **greenish-yellow discharge,** pelvic or swollen Bartholin glands. Men experience a **purulent** dysuria, and erythema of the urethral meatus.
- The differential diagnosis includes chlamydia, endom PID, vaginitis, UTI, salpingitis, and tubo-ovarian absce

### Diagnosis

- **Gram stain and culture** is the gold standard for any s urethra, or anus). **Nucleic acid amplification tests** ca vaginal tissue or from urine.
- Disseminated disease may present with **monoarticu** rash, and/or **tenosynovitis.** See Figures 2.8-23 and 2.8-

### Treatment

- **Ceftriaxone IM** and **azithromycin PO (regardless of is present).** Condoms are effective prophylaxis. Trea or partners if possible. Fluoroquinolones should not emerging resistance.
- Disseminated disease requires IV ceftriaxone for at leas





**sseminated gonococcal infection.** Hemorrhagic, painful pustules are
ses. (Reproduced with permission from Wolf Ket al. Fitzpatrick's Dermatology in General
McGraw-Hill; 2008.)

with pain; infertility; tubo-ovarian abscess with rupture;
occal infection (characterized by migratory polyarthralgia,
ustular skin lesions) (see Figure 2.8-24).

na pallidum, a spirochete. AIDS can accelerate the course
n.

**after infection):** Presents with a **painless ulcer** (**chancre;**
5A).
**after chancre):** Presents with low-grade fever, headache,
neralized lymphadenopathy with a diffuse, symmetric,
onpruritic) **maculopapular rash on the soles and palms**
25C, D). Highly infective 2° eruptions include mucous
**ylomata lata** (see Figure 2.8-26). Meningitis, hepatitis,
d eye involvement may also be seen.







F



G



H

**A)** Localized disease presenting with painless chancre and **(B)** dark-field microscopy **lis.** Maculopapular rash **(C)** [including palms and soles **(D)**] and **(E)** condylomata la as) in **3° syphilis. (G)** Rhagades (linear scars at angle of mouth), snuffles (nasal disch **in congenital syphilis.** (Part A reproduced with permission from the US Department of Health and Hum epartment of Health and Human Services and Renelle Woodall. Part C used with permission from Dr. Richard U lth and Human Services and Robert Sumpter. Parts E, H reproduced with permission from the US Department o mission from Chakir K, Benchikhi H. Centro-facial granuloma revealing a tertiary syphilis, Pan Afr Med J. 2013 Ju f Health and Human Services and Dr. Norman Cole.)



- **Early latent** (period from resolution of 1° or 2° sy the **first year** of infection): No symptoms; ⊕ serolog
- **Late latent** (period of asymptomatic infection **bey** No symptoms; ⊕ or ⊖ serology. One-third of cases lis.

- **3°** (late manifestations appearing 1–20 years after in sents with destructive, granulomatous **gummas. Neu tabes dorsalis** (posterior column degeneration), me **Robertson pupil** (constricts with accommodation l light). **Cardiovascular** findings include dilated aortic **root aneurysms,** and aortic regurgitation.

Diagnosis

confi matory tests.

**n:** Treat with benzathine penicillin. Give one dose for tion; give a weekly dose × 3 weeks for late latent infection atic infection of unknown duration.
Treat with penicillin IV × 10–14 days; penicillin-allergic e desensitized prior to therapy.

or a description of common sexually transmitted genital n outline of their diagnosis and treatment.

## inary Infections

FECTIONS

frequently than men, and ⊕ E coli cultures are obtained the mnemonic **SEEKS PP** for other pathogens. Risk fac-sence of catheters or other urologic instrumentation, ana- (eg, BPH, vesicoureteral reflux), previous UTIs or pyelo-nt antibiotic use, immunosuppression, and pregnancy.

**uria, urgency, frequency,** suprapubic pain, and hematu-

resent with **bedwetting,** poor feeding, recurrent fevers, g urine.
may present with delirium/acute confusion and few other

Remem
can resu
(headac
known a
**reactio**
release
organism

Genital
"do cry,"
The oth

Commo

**SEEKS**

**S**erratia
**E**coli
**E**nteroba
**K**lebsiella

| | | | |
|---|---|---|---|
| lation<br>ure | | | |
| ions<br>order. | Irregular, deep, well demarcated, necrotic. | Regular, red, shallow ulcer. | Irregular, pink or white, raised; caulifl wer. |
| | 1–3 | Multiple | Multiple |
| | 10–20 mm | 1–3 mm | 1–5 mm |
| | **Yes** | **Yes** | No |
| us | Inguinal lymphadenopathy. | Malaise, myalgias, and fever with vulvar burning and pruritus. | Pruritus. |
| biopsy<br>ies). | Difficult o culture; diagnosis is made on clinical grounds, culture on specialized media. | Tzanck smear shows multinucleated giant cells (best initial test; Figure 2.8-31); viral cultures (most accurate test); DFA or serology. | Clinical exam; biopsy for confi mation. |
| r | Azithromycin or ceftriaxone. | Acyclovir, famciclovir, or valacyclovir for 1° infection. | Cryotherapy, laser, or excision; topical agents such as podophyllotoxin, imiquimod, or |

urine culture.
nts with urosepsis should be hospitalized and initially **ntibiotics.** Consider broader coverage to include resistant coccus.
tibiotics may be given to women with uncomplicated Check for prostatitis in men.

---

nunity-acquired cases of pyelonephritis result from the same cystitis. Cystitis and pyelonephritis have similar risk factors.

oms are similar to those of cystitis but show evidence of **act disease.**
de **flank pain, fever/chills,** and nausea/vomiting. Dysuria, rgency are also possible.

**:** Results are similar to those of cystitis, but with **WBC** d cultures to rule out urosepsis.
ukocytosis.
eral, imaging is not necessary. Patients who relapse or do herapy within 48–72 hours should be evaluated by ultra- obstruction, abscess, and other complications.

patients may be treated on an outpatient basis for 7–14 **holones** are first-line therapy. Encourage ↑ PO fluids and

nister IV antibiotics to patients who have serious medical

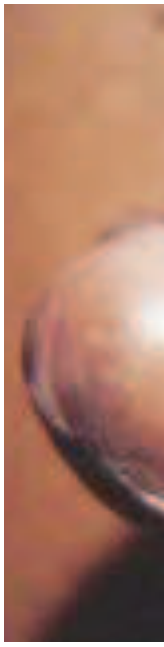


**FIGURE**
**tous pe**
duced with
Principles o
McGraw-H

Pyelone
serious
pregnan
untreate
develop



Mul-
ne labia.
Johnson
nopsis of
McGraw-

ing:
°C or
fever).
per
Hg on

90 bpm.
**ia:**
$m^3$
10%



the presumed site of infection).

- **IV drug users/indwelling lines:** S aureus, coagulas species.
- **Asplenic patients:** Pneumococcus, H influenzae, mer sulated organisms).

## History/Pe

- Presents with abrupt onset of fever and chills, altered m cardia, and tachypnea. Severe sepsis may lead to en such as renal or hepatic failure. **Hypotension** occur shock.
- **Septic shock** is typically a warm shock with **warm sk** This contrasts with cardiogenic shock, which typically **skin and extremities.**
- Petechiae, ecchymoses, or abnormal coagulation tests of cases).

## Diagnosis

- A clinical diagnosis.
- Labs show leukocytosis or leukopenia with ↑ bands (50% of cases), evidence of ↓ tissue perfusion (↑ creati tate), and abnormal coagulation studies (↑ INR).
- It is critical to obtain cultures of all appropriate sites CSF, wound, urine).
- Imaging (CXR, CT) may aid in establishing the etiol tion.

## Treatment

- ICU admission may be required. Treat aggressive **empiric antibiotics** (based on the likely source of i **pressors.**
- Treat underlying factors (eg, remove Foley catheter or
- The 1° goal is to **maintain BP** and **perfuse** end organs

s of the correct species is essential, because P falciparum is often resistant to standard chloroquine treatment.

**ght-stained thick and thin blood films** should be sent scopic evaluation to determine the species as well as the emia (see Figure 2.8-32).

monstrates normochromic, normocytic anemia with retic-

w, more sensitive serologic tests are available, including tection methods, fluorescent antibody methods, and PCR.

malarial infection can be treated orally. **Chloroquine** has d antimalarial medication, but increasing resistance often use of other medications.

ax, P ovale, or an unknown species, **primaquine** is added ypnozoites in the liver.

s can be treated with parenteral antimalarial medications with transition to oral regimens as tolerated. Symptoms ith supportive care.

**guanil and mefloquine are possible first-line chemopro- against chloroquine-resistant malaria.**

evere hemolytic anemia, renal impairment, noncardio- dema, hypoglycemia, lactic acidosis, acute hepatopathy,

NUCLEOSIS

**minata.**
lic Health I
and Preven

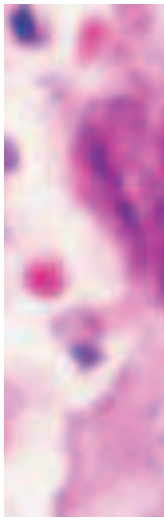


**FIGURE**
**genital l**
**ated gia**
Yale Rosen



**FIGURE**
falcipar
**thin sme**
**malaria.**
Public Hea
trol and Pr



**FIGURE 2.8-33. Pharyngitis.** (Used with permission from Dr. Richard Usatine

diagnosed with streptococcal pharyngitis (30% of pati
mononucleosis are asymptomatic carriers of GAS in th

- The differential diagnosis also includes **CMV,** toxopla
6, other causes of viral hepatitis, and lymphoma.

### Diagnosis

- Diagnosed by the **heterophil antibody (Monospot) te**
first few weeks after symptoms begin).
- **EBV-specific antibodies** can be ordered in patients w
nucleosis and a ⊖ Monospot test. **Infectious monon**
**that are Monospot ⊖ and EBV-antibody ⊖ are most**
**infection.** Acute HIV and other viral etiologies should
- CBC with differential often reveals mild **thrombocyt**
**lymphocytosis** and > 10% **atypical T lymphocytes.**
- Comprehensive metabolic panel usually reveals mildl
nases, alkaline phosphatase, and total bilirubin.

### Treatment

Treatment is mostly supportive, as there is no effective an
ticosteroids are indicated for airway compromise due to to
severe thrombocytopenia, or severe autoimmune hemolyt

### Complications

- **CNS infection:** Can present as aseptic meningitis, en

headache, myalgia, and malaise. The differential diagnowing:

endocarditis (eg, HACEK organisms; see the discussion of rditis), occult abscess (abdominal, prostatic), osteomyelictions, sinusitis. In HIV patients, consider MAC, histoplas-

phomas, leukemias, hepatic and renal cell carcinomas.
till disease, SLE, cryoglobulinemia, polyarteritis nodosa, disease, granulomatous disease (including sarcoidosis).
Pulmonary emboli/DVT, IBD, alcoholic hepatitis, drug editerranean fever, factitious fever.
5%).

sence of fever, and take a detailed history, including famal, occupational, dietary, exposures (pets/animals), and

CBC with differential, ESR, serum protein electrophoreod cultures, sputum Gram stain and culture, UA and culSpecific tests (ANA, RF, CK, viral cultures, viral serolo) can be obtained if an infectious or autoimmune etiology

a CXR. CT of the chest, abdomen, and pelvis should the workup of a true FUO. Invasive testing (marrow/liver ally low yield. Laparoscopy and colonoscopy are higher ne tests (after CT).

**medications.** Patients with FUO and a completely ⊖

Overall,
for the
(> 60%)
for □15
diseases

FUO pat
do not
therapy.

A 45-y
the ED
vomiti
She ha
and w
pyelon
bacter
in the
be a p
the ne
manag

# Tick-Borne Infections

## LYME DISEASE

- A tick-borne disease caused by the spirochete Borrelia
  seen during the **summer months,** and carried by Ixo
  tailed deer and white-footed mice. Endemic to the N
  Midwest, and Pacific coast.
- **Hx/PE:** Presents with the onset of rash with fever, ma
  ache, myalgias, and/or arthralgias. Infection usually
  feeds for > 18 hours.
  - **1° (early localized disease): Erythema migrans** b
    thematous macule or papule that is found at the t
    expands slowly over days to weeks. The border
    raised, often with central clearing ("bull's eye"; see
  - **2° (early disseminated disease):** Presents with mig
    thies, neurologic phenomena (eg, Bell palsy [bilate



alsy, radiculoneuropathy, carditis, heart block) + endemic
e serologies (IgM and IgG antibodies to B burgdorferi).
or serologic testing (must meet all criteria): endemic area
isk factor + symptoms consistent with early disseminated
se. Order ELISA IgM and IgG, if positive or equivocal,
n blot for confirmation. Do not use for "screening" or
ymptoms. Western blots sent without ELISA have high

**re/PCR:** Extremely difficult to obtain; not routinely done.

isease with **doxycycline** (or amoxicillin in children < 8
and in pregnant patients); more advanced disease (eg,
itic disease) should be treated with **ceftriaxone.**
piric therapy for patients with the characteristic rash,
r a tick bite acquired in an endemic area. Prevent with
dance.
Lyme disease is not usually transmitted within the first 48
f tick attachment. Give one dose of doxycycline if all the
ly: tick is Ixodes scapularis and has been attached for > 36
ylaxis is begun within 72 hours of removal, local rate of
icks with B burgdorferi is > 20%, and doxycycline is not
ed. If criteria not met, observe and only treat if erythema
lops.

## SPOTTED FEVER

by Rickettsia rickettsii and carried by the American dog
or variabilis). The organism invades the endothelial lining
causes **small vessel vasculitis.**
s with **headache, fever,** malaise, and **rash.** The charac-
itially macular (beginning on the wrists and ankles) but
ial/purpuric as it spreads centrally (see Figure 2.8-35).
atus or DIC may develop in severe cases.

Lyme di
vector-b

Lyme ar
minimal
and war

Rocky M
the wris
centrally

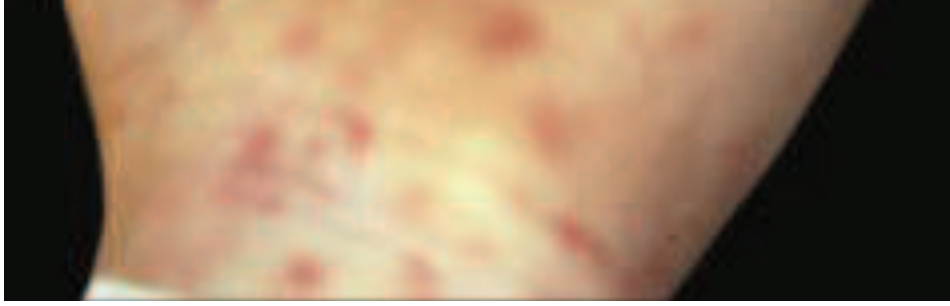


**FIGURE 2.8-35. Rocky Mountain spotted fever.** These erythema
evolve into a petechial rash that will spread centrally. (Reproduced with per
RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology, 7th ed. New York, N

# Infections of the Eyes and Ears

## INFECTIOUS CONJUNCTIVITIS

A common complaint in the ED setting, inflammation
is most often bacterial or viral but can also be fungal, p
chemical. It is essential to differentiate potentially visio
tious etiologies from allergic or other causes of conjunc
identify other vision-threatening conditions that may m
Table 2.8-11 lists the common etiologies of infectious con

## ORBITAL CELLULITIS

- Commonly due to direct spread of infection of the **pa**
  lead to endophthalmitis and blindness. Usually caus
  **staphylococci (including MRSA), and** H in*fl*uenza
  diabetic and immunocompromised patients, the Zygo
  Rhizopus must be included in the differential diagnosi
- **Hx/PE:** Presents with **acute-onset fever, proptosis, ↓**
  **ment,** ocular pain, and ↓ visual acuity. Look for a
  **trauma/surgery or sinusitis.** Palatal or nasal mucosal u

| | | |
|---|---|---|
| | involvement can lead to perforation and blindness. | intracellular diplococci. |
| | **Neonatal infection: mucopurulent conjunctivitis. Trachoma (global): recurrent epithelial keratitis** in childhood; trichiasis, corneal scarring, and entropion. **The leading cause of preventable blindness worldwide.** | Neonatal infection: Giemsa stain, chlamydial cultures. Trachoma: clinical (most often), PCR. |
| | **Viral** | |
| mmon) | Copious **watery discharge,** severe ocular irritation, preauricular lymphadenopathy. Occurs in epidemics. | |

of the external auditory canal, also known as "swimmer's
nas and Enterobacteriaceae are the most common etio-
h grow in the presence of excess moisture.
s with pain, pruritus, and possible purulent discharge.
eals **pain with movement of the tragus/pinna** (unlike oti-
n edematous and erythematous ear canal. See the Pediat-
discussion of otitis media.
iagnosis. Obtain a culture for severe or refractory cases.
if the patient appears toxic.

Diabetic
externa.

carditis in patients with a history of **IV drug abuse.**

- Viridans **streptococci:** The most common pathogen acute bacterial endocarditis and following **dental pr valves.**
- **Coagulase-⊖** Staphylococcus: The most common in **prosthetic valve** endocarditis.
- Streptococcus bovis: S bovis endocarditis is associated **malignancy.** Perform colonoscopy if S bovis diagnosed
- Candida **and** Aspergillus **species:** Account for most c carditis. Predisposing factors include long-term indw malignancy, AIDS, organ transplantation, and IV drug

### History/Pe

- **Constitutional symptoms are common (fever/F fatigue).**
- Examination reveals a **heart murmur.** The mitral va tation) is more commonly affected than the aortic va users; more right-sided involvement is found in IV d valve > mitral valve > aortic valve).
- **Osler nodes** (small, tender nodules on the finger and **lesions** (small peripheral hemorrhages; see Figure 2.8- **orrhages** (subungual petechiae; see Figure 2.8-36B), hemorrhages), focal neurologic deficits from emboli embolic phenomena are also seen.

**docarditis**

| Subacute | Marantic | Culture-⊖ (includes HACEK) | |
|---|---|---|---|
| dans streptococci | Cancer (poor prognosis). | Haemophilus | Lib |
| native valve, dental | Metastases seed valves; | parainfluenza | en |



B

**taneous manifestations of infective endocarditis. (A) Janeway** blization to the sole leads to a cluster of erythematous macules known **splinter hemorrhages.** The splinter hemorrhages shown along the late are due to emboli from subacute bacterial endocarditis. (Part A he Armed Forces Institute of Pathology, Bethesda, MD, as published in Knoop KJ et al. Atlas New York, NY: McGraw-Hill; 2002. Part B used with permission of the Department of Derma- al Center and Brooke Army Medical Center, San Antonio, TX, as published in Knoop KJ et al. ed. New York, NY: McGraw-Hill; 2002.)

factors, clinical symptoms, and the **Duke criteria** (see he presence of two major, one major + three minor, or ria all merit the diagnosis of endocarditis. Obtain serial om different sites before starting antibiotic therapy.
cytosis and left shift; ↑ **ESR** and CRP.

V **antibiotic treatment** for acutely ill patients. Vancomy- n is an appropriate choice for most patients. Tailor antibi-

**e Criteria for the Diagnosis of Endocarditis**

| Components |
| --- |

- The **preferred antibiotic prophylaxis** is amoxicillin. F penicillin allergic, use cephalexin, clindamycin, azithr mycin.

## ANTHRAX

Caused by the spore-forming gram-⊕ bacterium Bacill tion is an occupational hazard for veterinarians, farmers, a handle **animal wool, hair, hides, or bone meal produ** weapon. B anthracis can cause cutaneous (most commo deadly), or GI anthrax. **There is no person-to-person spre**

### History/Pe

- **Cutaneous:** Presents 1–7 days after skin exposure spores. The lesion begins as a **pruritic papule** that ulcer surrounded by a satellite bulbus/lesion with an a round, regular, raised edge. **Regional lymphadenop** teristic. The lesion evolves into a **black eschar** within ure 2.8-37).
- **Inhalational:** Presents with fever, dyspnea, hypoxia, hy toms of pneumonia (1–3 days after exposure), classic **rhagic mediastinitis.** Patients typically do not have pul
- **GI:** Occurs after the ingestion of poorly cooked, cont present with dysphagia, nausea/vomiting, bloody diarr pain.

### Diagnosis

Criteria for diagnosis include culture isolation or two no tests (PCR, immunohistochemical staining, or ELISA). C sitive test for inhalational disease (shows a **widened media** effusions).

the fore-
I. Steele,
ease
lished in
and Immu-
2010.)

limited motion of the adjacent joint. Systemic symptoms urulent drainage may be present.

ount; ↑ **ESR** and **CRP** levels in most cases. Blood cultures

en ⊖ initially but may show **periosteal elevation** within Bone scans are sensitive for osteomyelitis but lack specific-

t of choice) will show ↑ signal in the bone marrow and t tissue infection (see Figure 2.8-38).
agnosis is made by **bone aspiration** with Gram stain and **ical diagnosis made by probing through the soft tissue ually sufficient,** as aspiration carries a risk of infection.

**ement** of necrotic, infected bone followed by **IV antibi-**ks. Empiric antibiotic selection is based on the suspected ram stain.
mycin plus ciprofloxacin, ampicillin/sulbactam, or oxacil- methicillin-sensitive S aureus); vancomycin (for MRSA); ciprofloxacin (for gram-⊖ bacteria).

**mon Pathogens in Osteomyelitis**

| t hin K |
|---|
| Saureus. |
| Saureus or Pseudomonas. |

penetra
decubit

Diabetic
treated
gram-⊕

Penicilli
minimal
had an a
cephalo
a patien
to penic
antibiot



**FIGURE 2.8-38. Diskitis/osteomyelitis.** Sagittal contrast-enhance
tion of a lower thoracic intervertebral disk with abnormal enhancemen
cent vertebral bodies (arrows) and a posterior rim-enhancing epidural a
the spinal canal. (Reproduced with permission from Tintinalli JE et al. Tintinalli's Emergen
Study Guide, 6th ed. New York, NY: McGraw-Hill; 2004.)

## Complications

Chronic osteomyelitis, sepsis, septic arthritis. Long-standi
elitis with a draining sinus tract may eventually lead to **s**
**noma** (Marjolin ulcer).

pedic Injuries
erve Injuries
me
ne
236 Gout
238 Rheumatoid Arthritis
238 Seronegative Spondyloarthropat
ANKYLOSING SPONDYLITIS
239
240 Polymyositis and Dermatomyosi
241 Systemic Sclerosis
241 Systemic Lupus Erythematosus
241 Giant Cell Arteritis
242 Complex Regional Pain Syndrom
243 Fibromyalgia
244 Polymyalgia Rheumatica
244

| Presentation | Treatment |
| --- | --- |
| **dislocation:** Most common; risk of **axillary njury.** Patients hold arm in slight **abduction and l rotation** (see Figure 2.9-1).<br>**dislocation:** Rare; associated with **seizure and cution.** Patients hold arm in **adduction and rotation.** | Reduction followed by a sling and s dislocations may need surgical trea |
| ma. **Radial nerve palsy** may lead to wrist drop f thumb extension. | Hanging-arm cast vs coaptation spl Functional bracing. |
| **ft** fracture from direct trauma often in self- gainst a blunt object. | ORIF if significa tly displaced. |
| l fracture of the **proximal ulna** with **on of the radial head.** Results from fall on and outstretched arm. | ORIF of the shaft fracture and close radial head. |
| l fracture of the **radius** with **dislocation of the oulnar joint.** Results from a direct blow to the | ORIF of the radius and casting of th supination to reduce the distal radi |
| e **distal radius.** Often results from a **fall onto tched hand,** leading to a **dorsally displaced, ngulated fracture.** Commonly seen in the teoporosis) and children. | Closed reduction followed by appli cast; open reduction if the fracture |
| **monly fractured carpal bone. Results from a o an outstretched hand.**<br>weeks for radiographs to show fracture (see | Thumb spica cast. If displacement c present, treat with open reductio With proximal-third fractures, **AVN** |

2.9-3).

**nterior dislocation:** Can injure the **obturator nerve.**

---

risk with osteoporosis. Presents with a **shortened and externally rotated** leg.

an be radiographically occult, so a good clinical history with ⊖ radiographs warrants further evaluation with CT or MRI.

**isplaced femoral neck fractures** associated with an ↑ **risk of AVN** and nonunion.

ssociated with **DVTs.**

ORIF. Displaced femoral ne
may require a hip hemia

**Anticoagulate to ↓ the li**

Hip fracture involves the a
intracapsular femur.

---

irect trauma. Beware of **fat emboli,** which present ith fever, changes in mental status, dyspnea, hypoxia, etechiae, and ↓ platelets.

Intramedullary nailing of t
open fractures.

---

resent with knee instability and hematoma.

**CL:**

Result from a noncontact **twisting mechanism, forced hyperextension, or impact to an extended knee.**

⊕ anterior drawer and Lachman tests.

Rule out a meniscal or MCL injury (MCL injury = ⊕ valgus stress test; LCL injury = ⊕ varus stress test).

**CL:**

Result from a **posteriorly directed force on a fl xed knee** (eg, dashboard injury).

⊕ posterior drawer test.

**leniscal tears:**

Result from an **acute twisting injury or a degenerative tear** in elderly patients.

Clicking or locking may be present.

MRI is the diagnostic test c

Treatment of MCL/LCL and
**conservative.**

Treatment of ACL injuries i
**surgical** with graft from
tendons.

Operative PCL reconstruct
competitive athletes wit

Operative meniscal repair
reparable tears or older
symptoms who do not r
treatment.



A



B

**FIGURE 2.9-1. Anterior shoulder dislocation.** AP **(A)** and scapula
the right shoulder demonstrate anterior and inferior dislocation of the h
the glenoid. (Reproduced with permission from USMLE-Rx.com.)



**cture.**
nows
ist of
sion from



## Common Peripheral Nerve Injuries

Table 2.9-2 outlines the clinical findings of the most c
nerve injuries.

## Compartment Syndrome

↑ **pressure** within a confined space that compromises ner
tissue perfusion. Occurs primarily in the anterior compa
leg and in forearm 2° to trauma to the affected limb
injury). Also a concern with circumferential thermal inju
procedures.

### HisTory/Pe

- Presents with pain out of proportion to physical findi
**sive motion** of the fingers and toes; and **p**aresthesias,
mia, **p**ulselessness, and **p**aralysis (the **6 P's**).
- Paralysis and pulselessness occur as **late** signs of compa

B

**earm fractures. (A)** Monteggia; **(B)** Galeazzi; **(C)** Colles. (Parts A, B reproduced with permis
ew York, NY: McGraw-Hill; 2009. Part C reproduced with permission from Usatine RP et al., eds. The Color Atlas o

## unnel Syndrome

**median nerve at the wrist** caused by ↓ size or space of leading to paresthesias, pain, and occasionally paralysis. by overuse of wrist flexors; associated with diabetes mel- unction. Commonly occurs in pregnant and middle-aged

ning over the **thenar area of the hand** and proximal fore-

umbness is seen in a median nerve distribution.
**en at night,** when the wrists are held in flexion, or with se.
ows thenar atrophy (if CTS is long-standing).
**er** and **Tinel sign** are ⊕ (see Figure 2.9-5).

Open fra
emerge
the OR
and deb
repair. R
antibiot

**mon Peripheral Nerve Injuries**

| Motor deficit | Sensory deficit | Common causes |
|---|---|---|
| ension. | Dorsal forearm and hand (the first 3 fingers | Humeral fracture. Prolonged compression at level of humerus **("Saturday night palsy").** |



B

**el syndrome. (A)** Tinel sign. The Tinel test is performed by tapping the volar surface
uver. The Phalen maneuver is performed by compressing the opposing dorsal surface
n. This causes tingling over the median nerve distribution. (Reproduced with permission fro
dics: The Extremities, 5th ed. New York, NY: McGraw-Hill; 2007; as published in Tintinalli JE et al. Tintinalli's Emerg
Hill; 2010.)

leg.

d leg.
ally

nee
, and
**lateral**
nly

## Diagnosis

A clinical diagnosis, although nerve conduction studies a
be used to confirm.

## Treatment

- Splint the wrist in a neutral position at night and du
sible.
- Conservative treatment can include corticosteroid inj
canal.
- Surgical release of the carpal tunnel is a widely acce
ticularly for fixed sensory loss, thenar weakness, or i
with no improvement after splinting and/or glucocorti

## Complications

Permanent loss of sensation, hand strength, and fine moto

s

**condition** characterized by pain at tendinous insertions
l with swelling or impaired function. It commonly occurs
, **biceps,** wrist extensor, **patellar,** iliotibial band, posterior
**tendons.** Overuse is the most common cause.

ain at a tendinous insertion that worsens with **repetitive**
**d strength testing** of the affected muscle group.
ylitis (or tennis elbow) worsens with resisted extension of
edial epicondylitis with resisted flexion of the wrist.

s. Consider a radiograph if there is a history of trauma.
graphy may be useful in detecting tendon tears.

nd NSAIDs; apply ice for the first 24–48 hours.
ng, bracing, or immobilization.
ing exercises once pain has subsided.
treatment fails, consider corticosteroid injection. **Never**
**les tendon** in view of the ↑ risk of rupture. Avoid repeti-

## k Pain

e second-leading symptom-related cause for office visits in

Infection
occurs a
Infection
iatrogen
aspiratio

Oral fluo
with an
tendinit

Q

A 23-y
ED wit
right h
at a ba
of the
x-ray f
metac



**ion.**
lumbar
of the
on from
Medicine,

A

ure,
l
al

disk herniation (see Figure 2.9-6).
- Order a stat MRI for cauda equina syndrome or for a s
gressing neurologic deficit.
- Order an MRI if symptoms are refractory to conservati

### Treatment

- **NSAIDs** in scheduled doses, physical therapy, and loc
lution within 4 weeks in 80% of cases. Do not prescrib
**ation of regular activities** is preferred.
- Epidural injection or nerve block may be of benefit.
- Severe or rapidly evolving neurologic deficits and cau
are indications for surgical intervention.

## SPINAL STENOSIS

Narrowing of the lumbar or cervical spinal canal, leadin
the nerve roots and spinal cord. Most commonly due t
disease; typically occurs in middle-aged or elderly patients

### History/Pe

- Presents with neck pain, back pain that **radiates to th**
**tocks and legs,** and leg numbness/weakness.
- In lumbar stenosis, leg cramping is worse with standing
- In lumbar stenosis, symptoms **improve** with **flexion at**
ing forward, which relieves pressure on the nerves.

### Diagnosis

- Radiographs show degenerative changes that include
ing, facet hypertrophy, and sometimes spondylolisthes
rowed spinal canal.
- MRI or CT shows spinal stenosis.

| | | |
|---|---|---|
| gus), foot eversion muscles). | | aspect of the lower leg. |
| ion (gastrocnemius/ extension (gluteus | Achilles | Plantar and lateral aspects of the foot. |

## coma

nor, it is the most common primary malignancy of bone olescents. Tends to occur in the **metaphyseal** regions of **oximal tibia,** and proximal humerus; often metastasizes to osteosarcoma is often a result of sarcomatous transforma- tumors, including Paget disease.

gressive and eventually intractable **pain that worsens at**

ymptoms such as fever, weight loss, and night sweats may

nlargement over the site of the tumor may be seen.

ogy chapter for a discussion of osteosarcoma vs Paget dis-

show **Codman triangle** (periosteal new-bone formation l end of the lesion) or a **"sunburst pattern"** of the osteo- gure 2.9-7)—in contrast to the multilayered **"onion skin-** ssic for Ewing sarcoma and the **"soap bubble"** appear-

Red flag
weeks o
severe p
neurolo
sphincte

Bowel o
overflow
saddle-a
with cau
surgical

The mos
is osteo

A 55-y
of pro
LBP ar
exami
point
and ↓
the be



A



B

**FIGURE 2.9-7. Malignant bone tumors. (A) Osteosarcoma.** Fem
the typical "sunburst" appearance of osteosarcoma (arrows). **(B) Ewing**
teristic "onion skinning" of Ewing sarcoma (arrowhead) is evident in th
radiograph of the left hip. (Reproduced with permission from Kantarjian HM et al. MD
Oncology, 1st ed. New York, NY: McGraw-Hill; 2006.)

## Septic Arthritis

An infection of the joint space that can occur after open i
Prosthetic joints greatly ↑ the risk. Rheumatoid arthritis
(OA), and bacteremia from endocarditis and IV drug use
(see below).

### History/Pe

Presents as a warm, red, immobile joint. Palpable effusio
ent. Fevers and chills can be seen if the patient is bacteren

### Diagnosis

- The best test is **joint aspiration.** See Table 2.9-4.
- A **WBC count > 80,000 mm³,** ⊕ Gram stain, or ⊕ flu
- The most common organisms are Staphylococcus,
  gram-⊖ rods.

### Treatmen T

Empirically treat with **ceftriaxone and vancomycin** until

| | | |
|---|---|---|
| < 25 | < 25 | ≥ 50 |
| = serum | = serum | > 25 |
| | | (Crystal analysis for gout/ pseudogout) |

mm tory arthritis can become secondarily infected.

**us,** ↓ ROM, and initially **pain that worsens with activity but improves with rest.** Morning stiffness generally lasts iffness is also experienced after periods of rest ("gelling").

w **joint space narrowing,** osteophytes, subchondral scle- ondral bone cysts (see Figure 2.9-9). Radiograph severity e with symptomatology.
hows straw-colored fluid, normal viscosity, and a WBC ls/μL.
including inflammatory markers, are typically **normal.**

, **weight reduction,** and **NSAIDs.** Intra-articular cortico- s may provide temporary relief.

**arthritis vs Rheumatoid Arthritis**

| Osteoarthritis | Rheumatoid arthritis |
|---|---|
| ects the elderly; has slow onset | Af ects the young; presents with |

Classic f
10–20 y
"onion-s
diaphys
**wings** a

Classic f
**of bone**
presenti
along w
on x-ray
region c



**FIGURE 2.9-9. Osteoarthritis.** Plain radiographs show joint space and subchondral degenerative cysts involving the DIP and PIP joints, w
(Reproduced with permission from USMLE-Rx.com.)

- Consider **joint replacement** (eg, total hip/knee arthro cases.

## Gout

Recurrent attacks of **acute monoarticular arthritis** resulti lar deposition of **monosodium urate crystals** due to disor olism. Risk factors include male gender, obesity, postm women, and **binge drinking.**

### History/Pe

- Presents with excruciating joint pain of sudden onset.
- Most commonly affects the **first MTP joint** (podagr knees, ankles, and wrists; the hips and shoulders are ge
- Joints are erythematous, swollen, and exquisitely tende
- **Tophi** (urate crystal deposits in soft tissue) can be see ease (see Figure 2.9-10). Tophi can ulcerate and drain
- Uric acid kidney stones are seen with chronic disease



**phaceous gout.** Note the slowly enlarging nodule of the right second
holic, hypertensive man on hydrochlorothiazide. (Reproduced with permis-

**SAIDs** (eg, indomethacin) are first line. Colchicine may
but is inferior to NSAIDs.
used when NSAIDs are ineffective or contraindicated, as
se.
**erapy:**
for overproducers, those with contraindications to proben-
nt (tophi, renal stones, chronic kidney disease), and refrac-
**obenecid** for under-secreters.
an ↓ the incidence of **acute urate nephropathy.**
avoidance of triggers of hyperuricemia will prevent recur-
any patients. **Avoid alcohol consumption.**

Colchici
chemot
used ea
it can ca
suppres

## oid Arthritis

mune disorder characterized by chronic, destructive,
tis with **symmetric** joint involvement that results in syno-
d pannus formation, ultimately leading to erosion of adja-
e, and tendons. Risk factors include female gender, age
**R4.**

Diagnosis

- **Diagnostic criteria:**
  - ↑ **rheumatoid factor (RF)** (IgM antibodies against
    ence of **anti-CCP** antibodies.
  - ↑ ESR or CRP may also be seen.
  - Exclusion of acute, autoimmune, or crystalline cau
  - Inflammatory arthritis of three or more joints for >
- **Labs:**
  - Anemia of chronic disease is common.
  - Synovial fluid aspirate shows turbid fluid, ↓ viscos
    count (3000–50,000 cells/μL).
- **Radiographs:**
  - **Early:** Soft tissue swelling and juxta-articular demin
  - **Late:** Symmetric joint space narrowing and erosio
    11B).

Treatment

- **NSAIDs:** Can be ↓ or discontinued following succes
  **disease-modifying antirheumatic drugs (DMARDs).**
- DMARDs should be started early and include **methot**
  tial DMARD to start with), hydroxychloroquine, and
  ond-line agents include TNF inhibitors, rituximab (an
  nomide.





n the late teens and early 20s. Presents with fatigue, inter-
, and LBP that **worsens with inactivity and in the morn-**

⊕ Schober test), loss of lumbar lordosis, hip pain and stiff-
t expansion are seen as the disease progresses.
and **heart block** can occur.
enthesitis (pain at insertion of tendons/ligaments) at the

seronegative spondyloarthropathy must be ruled out,
lowing:
**ritis** (formerly known as **Reiter syndrome**): A disease of
The characteristic arthritis, uveitis, conjunctivitis, and ure-
follow an infection with Campylobacter, Shigella, Salmo-
ydia, or Ureaplasma.
**hritis:** An oligoarthritis that can include the **DIP joints.**
th psoriatic skin changes and **sausage-shaped digits** (dac-
films show a classic "pencil in cup" deformity.
**spondylitis:** An ankylosing spondylitis–like disease char-
sacroiliitis that is usually asymmetric and is associated with

ound in 85–95% of cases.
y show **fused sacroiliac joints,** squaring of the lumbar ver-
ment of vertical syndesmophytes, and **bamboo spine** (see

in 75% of cases.
clear antibody (ANA).

domethacin) for pain; exercise to improve posture and

Reactive
can't pe
(arthritis



prominences.

History/Pe

- Patients may also develop myocarditis and cardiac con
- Can be associated with an underlying malignancy, breast carcinoma.

Diagnosis

- ↑ **serum creatine kinase** and **anti-Jo-1 antibodies** car 2.9-8).
- Diagnosis is generally made based on characteristic cli presentation without tissue biopsy; however, additiona ful for atypical presentations.

Treatment

- High-dose corticosteroids with taper after 4–6 weeks to dose.
- Azathioprine and/or methotrexate can be used as steroi

## Systemic Sclerosis

Also called scleroderma; characterized by inflammation t sive tissue fibrosis through excessive deposition of types Commonly manifests as **CREST syndrome** (limited form



| | |
|---|---|
| | Drug-induced SLE |
| | Polymyositis/dermatomyositis |
| | Primary biliary cirrhosis |
| | Systemic sclerosis |
| | SLE |
| | Autoimmune hepatitis |
| | Systemic sclerosis |
| | Graves disease |
| | Vasculitis, especially Wegener |
| | Vasculitis, microscopic polyangiitis |
| | RA |
| | Mixed connective tissue disease |

nvolving the skin as well as the GI, GU, renal, pulmo-
cular systems. Risk factors include female gender and age

y reveal symmetric thickening of the skin of face and/or



CREST s

Calcinos
Raynaud
Esophag

- **Calcium channel blockers** for Raynaud phenomenon
- **Angiotensin-converting enzyme inhibitors** for renal vention/treatment of renal crisis.

### Complications

Mortality is most commonly due to complications of p sion, as well as resulting renal or cardiac disease.

## Systemic Lupus Erythematosus

A multisystem autoimmune disorder related to antibod attack and deposition of antigen-antibody complexes. E highest risk. Usually affects women of childbearing age.

### History/Pe

- Presents with nonspecific symptoms such as fever, ar and symmetric joint pain.
- The mnemonic **DOPAMINE RASH** summarizes th nosing SLE (see also Figure 2.9-14). Patients with fou likely to have SLE (96% sensitive and specific).

### Diagnosis

- A ⊕ ANA is highly sensitive but not specific. **Antibo Sm** are highly specific but not as sensitive (see Table 2
  - **Drug-induced SLE:** ⊕ antihistone antibodies ar **cases** but are nonspecific.
  - **Neonatal SLE:** Associated with ⊕ anti-Ro antibod mother to neonate.
- The following may also be seen:
  - Antiphospholipid antibodies.

**s,** and **jaw claudication.**
t **monocular blindness,** weight loss, and myalgias and/or
cially of the shoulders and hips) are also seen.

r. (Influenced by age).
evaluation.
**biopsy: Definitive test.** Look for thrombosis; necrosis of
ymphocytes, plasma cells, and giant cells.

**prednisone** immediately to prevent ocular involvement
of the remaining eye after onset of monocular blindness).
ribution to vision loss, give **pulse-dose steroids.**
, but do not delay treatment. Conduct a follow-up eye

nifestation is blindness 2° to occlusion of the central reti-
of the internal carotid artery) that may initially present as

## Regional Pain Syndrome

ccompanied by **loss of function and autonomic dysfunc-**
ring after trauma. The disease has three phases: acute/
phic phase → atrophic phase. Not linked with true nerve

**curs** out of proportion to the initial injury, often in a **non-**

**Libman**
Noninfe
on the r
SLE and

SLE can

SLE and
PIP joint
nondefo

- A chronic pain disorder characterized by soft tissue an in the absence of joint pain. Inflammation is notabl 2.9-9). May be difficult to distinguish from myofascial areas).
- **Hx/PE:** Most common in **women 30–50 years of a** depression, anxiety, sleep disorders, inflammatory bo cognitive disorders ("fibro fog").
- **Dx:** Multiple (≥ 11 of 18) painful areas over all four the axial skeleton must be present for diagnosis. The 18 painful areas or non-fibromyalgia-associated painfu **myofascial pain syndrome.**
- **Tx: Antidepressants** (a selective serotonin reuptak antidepressant combination and two serotonin-norep inhibitors [SNRIs] have proven efficacy), gabapentin, relaxants, and **physical therapy** (stretching, heat app apy). Avoid narcotics. Multidisciplinary patient educat

## Polymyalgia Rheumatica

An inflammatory rheumatic condition characterized by in the shoulders, hips, and neck. Associated with **tempor** tors include female gender and age > 50 (see Table 2.9-9)

**TABLE 2.9-9. Fibromyalgia vs Polymyalgia Rheumatica**

| Characteristic | Fibro Myalgia | polyM |
|---|---|---|
| Age and sex | Middle-aged women (30–50 years of age). | Women > 5 |

y 258

258

258

AGE 263

AGE 263

HEMATOMA 264

BOSIS 264

266

IC HES 267

268

268

270

271

TIONAL VERTIGO 271

LOPATHY (LABYRINTHITIS OR VESTIBULAR NEURITIS) 272

272

273

muscular Junction 274

274

ENIC SYNDROME 274

275

ME 276

LEROSIS 276

277

277

279

FRONTOTEMPORAL DEMENTIA (PICK DISEASE)

NORMAL PRESSURE HYDROCEPHALUS

CREUTZFELDT-JAKOB DISEASE

LEWY BODY DEMENTIA

Movement Disorders

HUNTINGTON DISEASE

PARKINSON DISEASE

Neoplasms

Neurocutaneous Disorders

NEUROFIBROMATOSIS

TUBEROUS SCLEROSIS

Aphasia

BROCA APHASIA

WERNICKE APHASIA

Coma

Nutritional Deficiencies

Ophthalmology

VISUAL FIELD DEFECTS

GLAUCOMA

CATARACTS

AGE-RELATED MACULAR DEGENERATION

RETINAL VASCULAR OCCLUSION

Disruption of cerebral blood flow leads to death of brai
acute onset of focal neurologic deficits. Can be ischem
rhagic (20%). Table 2.10-5 contrasts modifiable and non
tors associated with stroke. Common etiologies are listed b

- **Atherosclerosis** of the **extracranial** and **intracrania**
  common carotid, basilar, and vertebral arteries).
- **Chronic hypertension, hypercholesterolemia,** or **di**
  perforating vessels supplying **deep** regions of brain,
  **infarcts.**
- **Cardiac or aortic emboli.**
- **Other causes:** Hypercoagulable states, craniocervical
  sinus thrombosis, sickle cell anemia, vasculitis (eg, gian

### History/Pe

Symptoms are dependent on the vascular territory affected

### Diagnosis

- **Emergent head CT without contrast** (see Figure 2.
  ate ischemic from hemorrhagic stroke and identify pot
  **thrombolytic therapy. Ischemic strokes < 6 hours old**
  **ible on CT scan.** Labs to draw immediately, in case th
  or intervention may be required, include CBC, PT/PT
  and troponin, BUN/creatinine.
- **MRI** (see Figure 2.10-1B) to identify early ischemic c
  on CT.

**TABLE 2.10-1. Spinal Tract Functions**

| Tract | Function | Clinica |
|---|---|---|

| | |
|---|---|
| | of cervical region. |
| Amyotrophic lateral sclerosis | Combined UMN and LMN deficits with no sen<br>both UMN and LMN signs.<br>Commonly presents as fasciculations with eve<br>hands; fatal.<br>Riluzole treatment modestly ↑ survival by ↓ p<br>Commonly known as Lou Gehrig disease in th<br>neuron disease in the United Kingdom. For<br>rilouzole. |
| Complete occlusion of anterior spinal artery | Spares dorsal columns. |
| Tabes dorsalis | Caused by 3° syphilis. Results from degenerat<br>columns and roots → impaired sensation a<br>sensory ataxia (inability to sense or feel the<br>Associated with Charcot joints, shooting pain<br>Exam will demonstrate absence of DTRs and |
| Syringomyelia | Syrinx (CSF-filled c vity within spinal cord) ex<br>white commissure of spinothalamic tract. Can<br>seen in 35% of Chiari malformations. Results i |

complication in AIDS, Lyme disease, Sarcoidosis, Tumors, and
es.

tract
(UMN
lesion =
Central
facial)

Facial
nucleu



T et al. First Aid for the USMLE Step 1 2015. New York, NY: McGraw-Hill; 2015.

Ges—

oss in

f the

- Determine underlying cause of stroke:
  - Cardioembolic: **ECG; echocardiogram.**
  - Thrombotic: Carotid ultrasonography; MRA; CTA
    pler; conventional angiography (see Figure 2.10-1C
  - Other potential causes that should be worked up if
    of suspicion: hypercoagulable states; sickle cell disea

Tr ea Tmen T

**Acute treatment measures** are as follows:

- **Hemorrhagic stroke:** See the discussion of intracerebr
- **Ischemic stroke:**
  - **Thrombolytics (tPA)** if it has been < **3 hours** since
    there is no bleeding or absolute contraindications

**TABLE 2.10-4. Commonly Tested Refl xes**

drugs)

ved in stroke for perfusion of ischemic area, but patient's
ust be < 185 and diastolic BP < 110 mm Hg for tPA.
been > 3 hours since onset of stroke. Consider switching
l in patients who are already taking ASA.
**nd hyperglycemia** as both are associated with worse prog-
etting of acute stroke.
signs and symptoms of brain swelling, ↑ ICP, and hernia-
utely with **mannitol** and **hyperventilation.**
treat poststroke complications such as aspiration pneumo-
l DVT.

**g-term treatment measures:**

**f hypertension (single greatest risk factor for stroke),**
**lemia,** and **diabetes.**
**grel.**
**:** In new atrial fibrillation (AF) or hypercoagulable states,

**mon Stroke Symptoms by Vessel Territory**

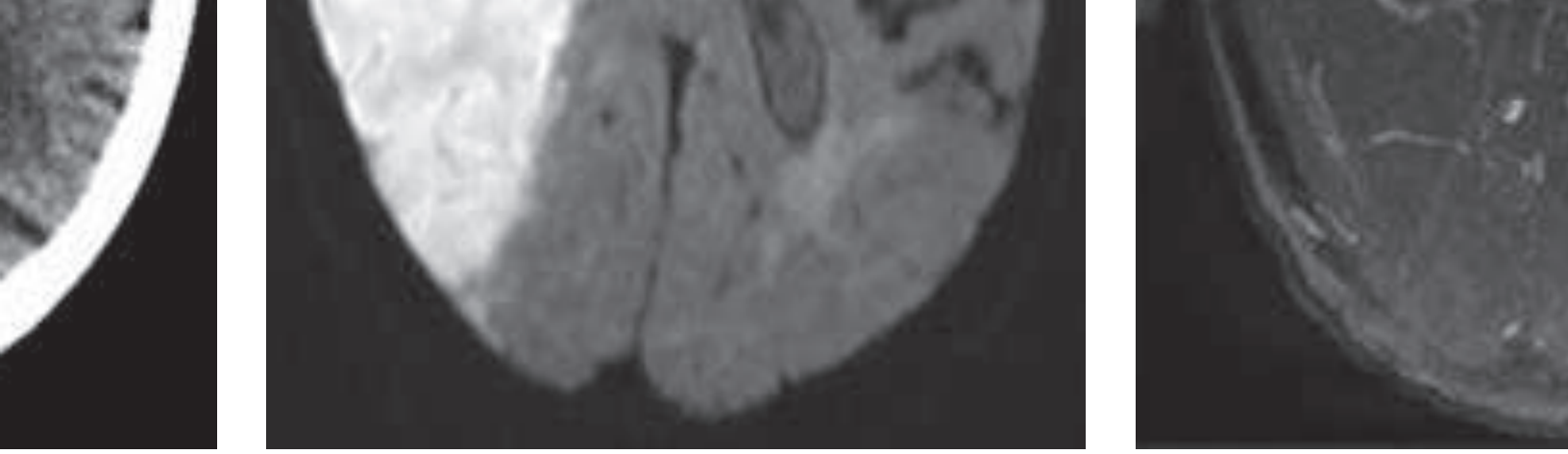
| d iSt ing u iSh ing SyMpt o MS |
|---|
| **ateral paresis** and **sensory loss** in the **face** and **arm;** gaze preference |
| **rd** the side of the lesion. |
| **minant hemisphere—neglect.** |
| **nt hemisphere—aphasia.** |

Contra
(major

**SAMPI**

**S**troke or
3 mon
***A**nticoa
prolon
**M**I in pas
**P**rior intr
**L**ow plat
***E**levate
> 110
Major **S**u
**T**IA (mild
of syn
**G**I or urin
**G**luco
***E**levate
blood
**S**eizures

*If values
treatment
tPA treatm

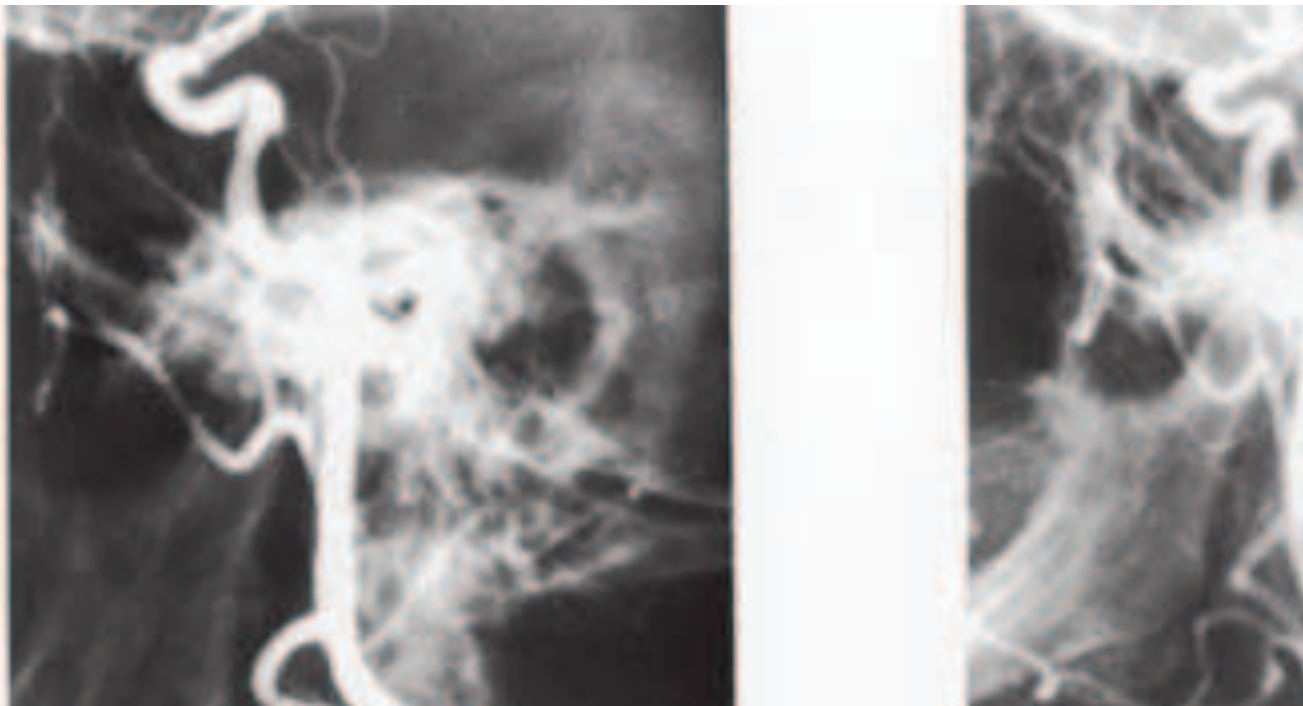


B C

**emic stroke.** Acute left hemiparesis in a 62-year-old woman. **(A)** Noncontrast head C
and asymmetrically decreased size of the right lateral ventricle in a right MCA distrib
RI with reduced diffusion in the same distribution, consistent with an acute infarct;
**modality for diagnosing an acute ischemic infarct. (C)** MRA shows the cause: an
Compare with the normal left MCA (yellow arrowhead). (Reproduced with permission from

the target INR is 2–3. In cases involving a prosthetic v
is 2.5–3.5.

- **Carotid endarterectomy:** If stenosis is > 60% in symp
> 70% in asymptomatic patients (contraindicated in
Figure 2.10-2).



e seen.
hird of patients will give a history of a **"sentinel bleed"** days to weeks prior to presentation.
of neurosurgical intervention, rapid development of ocephalus or seizures often leads to ↓ arousal or frank

**CT without contrast** (see Figure 2.10-3A) to look for arachnoid space.
o look for **RBCs, xanthochromia** (yellowish CSF due to BCs), ↑ protein (from the RBCs), and ↑ ICP.
**ography** (or equivalent noninvasive angiography such as with 3D reconstructions) should be performed once SAH ned (see Figure 2.10-3B–D) to identify source of bleeding. iography is warranted in high-risk cases and in those with picion even if CT and LP are unrevealing.

**definitive treatment!** May perform angiographic coiling o stabilize aneurysm.
**ing** (most likely to occur in the first 24 hours) by main- BP < 150 mm Hg until the aneurysm has been clipped or

**sm and subsequent ischemic stroke** (most likely to occur SAH) by administering calcium channel blockers (CCBs), **ine.**
**major** cause of **delayed morbidity and mortality** in H.
the head of the bed and instituting hyperventilation in an 30 minutes after onset).
**halus** through a lumbar drain, serial LPs, or ventriculo-

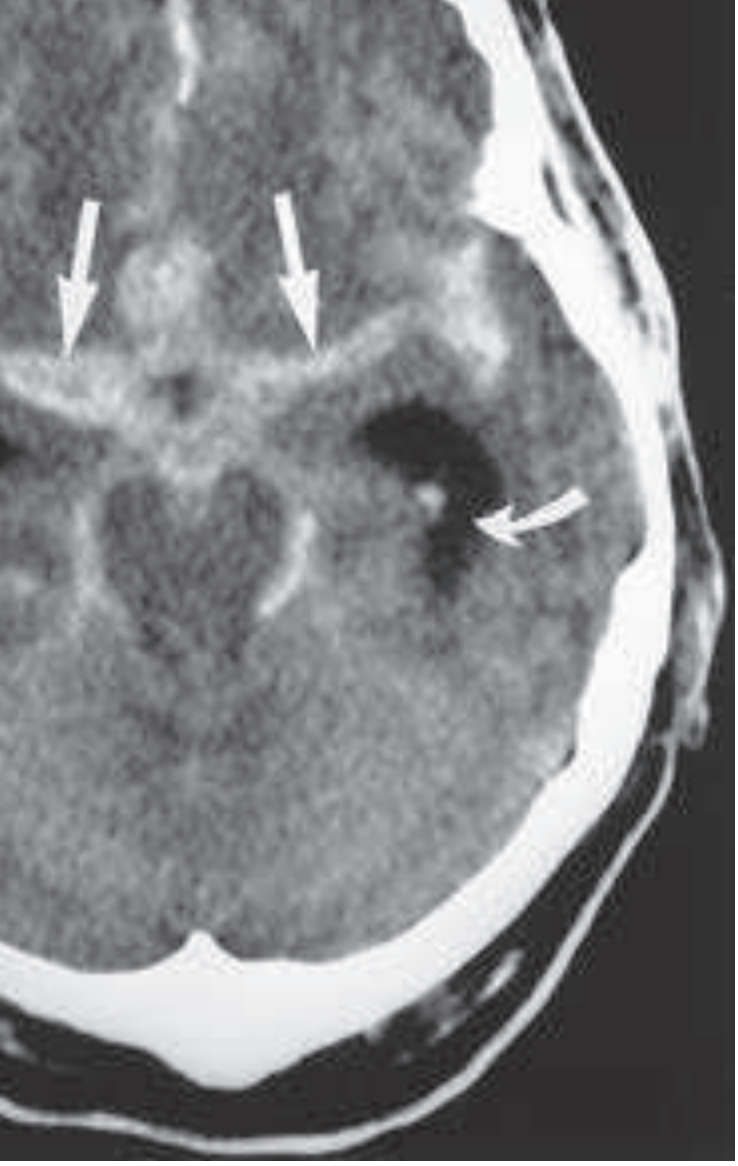
gradual
intensity

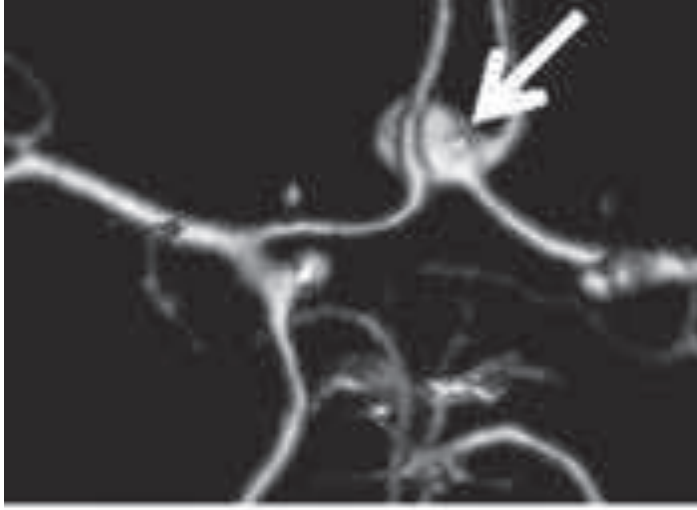
Conditi
aneury
more li

**M**arfan s
**A**ortic co
**K**idney d
polyc
**E**hlers-D
**S**ickle ce
**A**therosc
**H**istory (
**H**yper

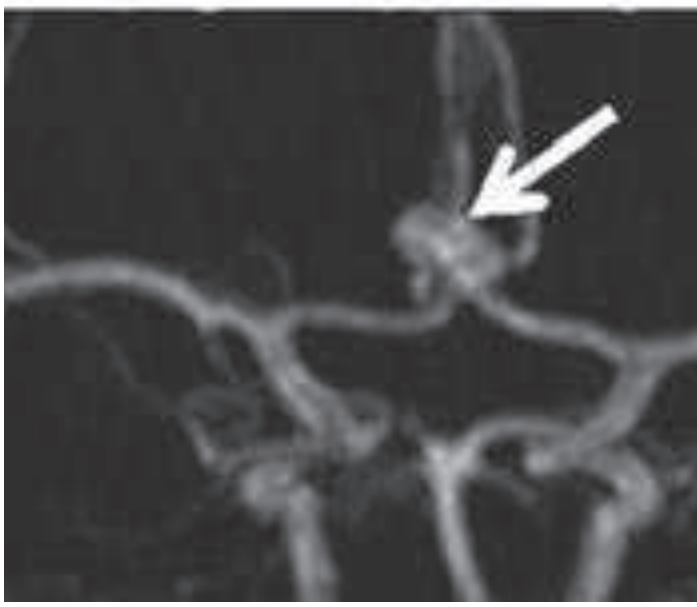
Q

A 68-y
ED wit
right f
numb





C



D

**oid hemorrhage.** Noncontrast CT **(A)** showing SAH filling the basilar cisterns and s
the dilated temporal horns of the lateral ventricles/hydrocephalus. Coned-down ima
am **(C),** and an MRA **(D)** show a saccular aneurysm arising from the anterior commu
ntinalli JE et al. Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 6th ed. New York, NY: McGraw-Hill;
Diagnosis & Treatment: Surgery, 13th ed. New York, NY: McGraw-Hill; 2010.)

A

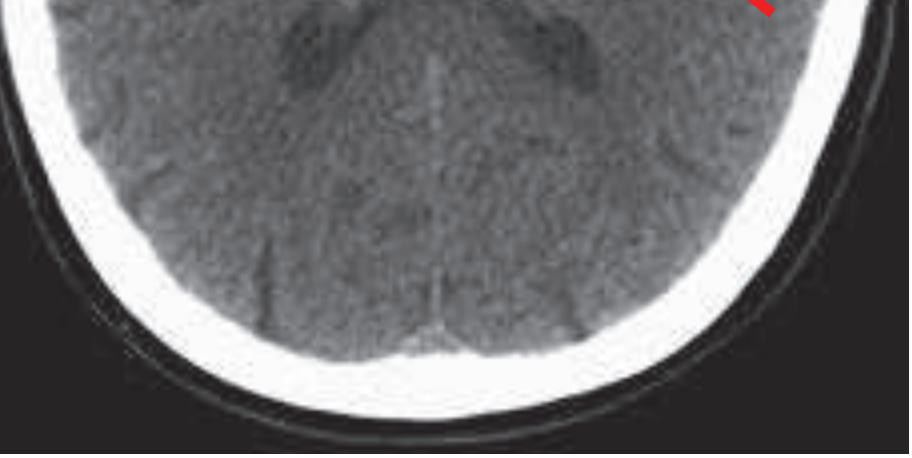
on,
sory
e

## Diagnosis

Immediate noncontrast head CT (see Figure 2.10-4). L
areas, mass effect, or edema that may predict herniation.

## Treatment

- Similar to that of SAH. Monitor for signs of herniation
  - Suspect herniation if patient develops Cushing
    bradycardia, irregular respirations), fixed pupils, c



**tracerebral hemorrhage.** Noncontrast head CT shows an intrapa-
(H) and surrounding edema (arrows) centered in the left putamen, a
pertensive hemorrhage. C, P, and T denote the normal contralateral
halamus. (Reproduced with permission from Fauci AS et al. Harrison's Principles of Internal
: McGraw-Hill; 2008.)

Mental s
an expa
occur w
and clas
of consc
→ gradu
With a s
changes
weeks.

**dural Hematoma vs Epidural Hematoma**

| Subdural | E |
|---|---|
| Head trauma → rupture of **bridging veins** → accumulation of blood between dura and arachnoid membranes. | Head trauma → lateral sk **meningeal artery** → acc skull and dura mater. |
| Elderly, alcoholics. | Severe trauma. |
| Headache; changes in mental status; contralateral hemiparesis; focal neurologic findings; me tal status changes in elderly.<br>Onset: subacute or chronic. | Immediate loss of consci interval (minutes to hour |
| CT findings<br>**Crescent-shaped, concave hyperdensity acutely (isodense subacutely; hypodense chronically)** (see | CT findings<br>**Lens-shaped, biconvex** 2.10-5B). |



A B

**FIGURE 2.10-5. Subdural vs epidural hematoma. (A) Subdural** crescent shape and the mass effect with midline shift. **(B) Epidural he** biconvex lens shape. (Reproduced with permission from Aminoff MJ. Clinical Neurology &Lange; 1996.)

sinus. Staphylococcus aureus is the most common causa antimicrobials have greatly ↓ both incidence and mortalit

### History/Pe

- **Headache is the most common presenting symptom.**
- Patients may present with orbital pain, edema, visual oculomotor, abducens, or trochlear nerve involvemen they typically appear ill and have a **fever.**
- **Late findings:** Changes in mental status such as confu coma suggest spread to the CNS or sepsis.

### Diagnosis

- **MRI** (with gadolinium and MR venography) is main n but CT angiography and CT venography are also often
- Lab studies show ↑ WBC count.
- Blood cultures reveal the causative agent in up to 50%

### Treatment

- Treat aggressively and empirically with broad-spectrum
  - **Penicillinase-resistant penicillin** (nafcillin or oxa **or fourth-generation cephalosporin** (eg, ceftriaxon

ruptions in normal sleep patterns.

throbbing headache (> 2 hours but usually < 24 hours) with **nausea, vomiting, photophobia,** and **noise sensitiv-** usually relieved by **sleep and darkness.** Migraine can be **aura.**

**ntation of migraine with aura: Unilateral pulsating HA,** a visual **aura** in the form of either scintillating scotomas hing lights) or visual field cuts.

and an otherwise ⊖ workup.

**ggers.**

**y** includes **triptans** (after OTC NSAIDs have failed), ion to other analgesics such as naproxen. Consider symp- t for nausea.

frequent or severe migraines includes **anticonvulsants** (eg, ramate), **TCAs** (eg, amitriptyline), **β-blockers** (proprano-

ore often than women; average age of onset is 25.

rief, **excruciating, unilateral periorbital headache** that to 3 hours, during which time the patient tends to be s.

ccur in **clusters,** affecting the same part of the head at the y (commonly during sleep) during a certain season of the

If a 25-y
repeate
unilater
with ips
headach

If a 30-y
headach
worsen
relaxatio
type hea

Q

A 28-y
prior m
throbb
is exac
minim
and ly
would
provid
What
prescr

most
nosed

nset of
rologic
norning
er 50
gns or
che

A

ches.
n for

### Diagnosis

Must have at least two of the following characteristics pressing/tightening quality; mild-moderate intensity; not tine physical activity. Rule out giant cell arteritis in patien with new headaches by obtaining an ESR, even if headach associated constitutional or vascular symptoms.

### Treatment

Relaxation, massage, hot baths, and **avoidance of ex NSAIDs** and **acetaminophen** are first-line abortive thera also be considered.

## SECONDARY HEADACHES

Consider secondary headaches when "red flags" are presen

### History/Pe

- Significant findings include **fever or rash** (consider infectious causes), **jaw claudication** (specific for te constitutional symptoms such as **weight loss** (associa inflammatory, or infectious conditions).
- **Photophobia, nausea, vomiting, and neck stiffnes** with aneurysmal SAH and meningitis due to meningea
- Conduct full general and neurologic exams, including a
- **Neurologic sequelae:** Look for **diplopia,** mental stat ciated symptoms (numbness, weakness, dizziness, a bances), **papilledema,** or pupillary abnormalities (par Horner syndrome).

### Diagnosis

- **If SAH is suspected,** obtain a **head CT without contr**

Brain tumor

ge. Etiologies of seizures, and their distinguishing features,
g:

sy (recurrent, unprovoked seizures): May be due to genet-
tal factors, early life brain injuries, etc.
y could be caused by the following:
ain lesion (tumor, stroke, AVM hemorrhage, or develop-
mality). Tend to have **focal** onset or focal postictal deficit,
**cal CNS pathology.**
gic etiologies: **Hypoglycemia, hyponatremia, hypocal-
rosmolar states, hepatic encephalopathy,** uremia, por-
**overdose** (cocaine, antidepressants, neuroleptics, methyl-
locaine), **drug withdrawal** (alcohol and other sedatives),
yperthermia, hypertensive encephalopathy, head trauma,
hypoperfusion.

ed to determine whether seizure is **partial, generalized,** or
ilepticus will be discussed in a separate section.

al electrical activity arises from a **discrete region (or multi-
ons) of the brain.** Can involve motor features as well as sen-
or psychic features (eg, fear, déjà vu, hallucinations). **Aura**
itory, visual, olfactory, or tactile hallucinations). A postictal
deficit (eg, **hemiplegia/hemiparesis,** or **Todd paralysis**) is
ally resolves within 24 hours. Can be **simple** or **complex.**
**mpaired level of consciousness.**
pically involve the **temporal lobe** (70–80%) with bilateral
aberrant electrical discharge, **leading to impaired level
ness.** Postictal confusion/disorientation/amnesia are char-

If a patie
twitchin
aware o
partial s

Both sim
seizures
seizures

If a patie
of lip sm
impaired
followed
partial s

- **Partial seizures:** Look for epileptogenic focus.
- **Childhood absence epilepsy:** EEG shows 3-pe wave discharges (remember classic EEG findings about learning how to read them!). EEG changes **hyperventilation.**
- **Tonic-clonic:** EEG typically shows 10-Hz activi phase and slow waves during the clonic phase.
- If EEG is normal during event concerning for sei **doseizures.**

- Serum prolactin levels may be elevated in the immed of generalized and complex-partial seizures.
- Rule out systemic causes with a CBC, electrolytes, c cose, LFTs, a renal panel, RPR, ESR, and a toxicology
- A focal seizure implies a focal brain lesion. Evaluate contrast.

## Tr ea Tmen T

- **Secure airway** when appropriate.
- For acute seizures lasting **longer than 5 minutes, see epilepticus below.**
- In cases of systemic 2° seizures, **treat the underlying c**
- For **partial** and **tonic-clonic** seizures, levetiracetam ( carbamazepine (Tegretol), phenobarbital, and valpro efficacy and can be used as chronic monotherapy.
  - In **children, phenobarbital** is the first-line anticonv
  - If a certain antiepileptic is ineffective as monothe tive. If alternative is ineffective, try a regimen of tics.
  - Other treatment options include gabapentin, top bazepine.
- **Absence seizures: First line is ethosuximide;** second l
- **Intractable temporal lobe seizures:** Consider **anteri tomy.**

**, breathing, circulation (ABCs);** consider rapid intuba-
rotection.
potential etiologies, administer **thiamine, followed by**
**oxone.**
ategies vary greatly among different institutions. Following
ide:
**enzodiazepine** (lorazepam or diazepam) within 0–5 min-

dose of IV benzodiazepine at 5–10 minutes.
still seizing at 20 minutes, give **fosphenytoin,** valproate
nobarbital, levetiracetam, or continuous infusion of mid-

y status epilepticus (RSE), give continuous antiepilep-
Ds) with bolus for breakthrough SE. Early intubation is
en giving continuous AEDs.

nditions that cause vertigo, it is worth defining **vertigo** and
m **lightheadedness.** Many times, **"dizziness"** is used to
lightheadedness. **Vertigo** is feeling like **you or your sur-**
**ng,** though in actuality there is **no movement,** while **light-**
one complains that they are about to **faint** or **"pass out."**

MAL POSITIONAL VERTIGO

f recurrent **peripheral vertigo** resulting from an **otolith**
ds to disturbances in the semicircular canals.

Though
tradition
> 30 mi
for any s
prevent
toxicity.

**Withdr**
seizures
**A**lcohol
**B**enzodi
**B**arbitur
**A**nticon

### History/Pe

- Presents with **acute onset of severe vertigo, head m and gait unsteadiness** accompanied by **nausea, vom mus.**
- **Labyrinthitis:** Auditory or aural symptoms (tinnitus, e ing loss). Lateral pontine/cerebellar stroke (anterior artery territory) may present with similar symptoms.
- **Vestibular neuritis:** Lacks auditory or aural symptoms cerebellar stroke (posterior inferior cerebellar artery te with similar symptoms.

### Diagnosis

- A **diagnosis of exclusion** once the more serious cause ebellar stroke) have been ruled out.
  - Acute peripheral vestibulopathy demonstrates the fo
    - An abnormal vestibulo-ocular reflex as determ head impulse test (ie, rapid head rotation fro while staring at the examiner's nose).
    - A predominantly horizontal nystagmus that a direction, opposite the lesion.
    - No vertical eye misalignment by alternate cover
  - If patients are "high risk"—ie, if they have atypical rologic symptoms or signs, cannot stand independ neck pain, are > 50 years of age, or have one or m tors—MRI with diffusion-weighted imaging is indic

### Treatment

Acute treatment consists of corticosteroids given < 72 h onset and antivertigo agents (eg, meclizine). The conditi spontaneously within weeks to months.

y changes that limit salt intake to avoid fluid retention;

teral cases, intratympanic injection of gentamicin into the rbed by the inner ear) has been shown to reduce the fre- rity of vertigo attacks.

**ommon causes** of loss of consciousness **2° to a sudden rfusion.** Etiologies can be **cardiac** or **noncardiac.** Presyn- ted with noncardiac causes and is described as a feeling of nsciousness without actual fainting. Commonly **confused**

ort a **trigger** (eg, orthostatics, standing for a long period of f blood, Valsalva maneuver).
pically involves prodromal symptoms that lead to loss of d muscle tone for < 30 seconds and recovery within sec- the following potential etiologies:
**hypotension:** Symptoms triggered by postural changes. uses include dehydration and autonomic neuropathy een in patients with diabetes).
udden increase in vagal tone triggered by prolonged stand- al distress, painful stimuli. Patient may experience prodro- s of nausea, warmth and diaphoresis prior to syncope.
not typically involve prodromal/postdromal symptoms; e history of arrhythmia, valvular disease, structural heart or the following potential etiologies:
**lar outflow tract obstruction (LVOTO)** (eg, **aortic ste- rophic obstructive cardiomyopathy**); syncope with exer- exercise

# Disorders of the Neuromuscular Junct

## MYASTHENIA GRAVIS

An **autoimmune disorder** caused by antibodies that b
**acetylcholine (ACh) receptors** located at the neuromusc
often affects young adult women, and can be associated
**rotoxicosis,** and other autoimmune disorders.

### History/Pe

- Presents with fluctuating **ptosis or double vision,** bu
  dysarthria, dysphagia), and **proximal muscle weakne**
  **cally worsen at the end of the day.**
- Patients may report difficulty in: climbing stairs, rising
  ing hair, and swallowing.
- **Myasthenic crisis** is rare but includes the potentially
  of respiratory compromise and aspiration.

### Diagnosis

- Best initial diagnostic test is acetylcholine receptor
  nium (Tensilon test) is an anticholinesterase that can b
  tic tool because it rapidly reverses symptoms.
- An abnormal **single-fiber EMG** and/or a **decremental**
  **tive nerve stimulation** can yield additional confirmatio
- **Chest CT** is used to evaluate for thymoma.

### Treatment

- Anticholinesterases **(pyridostigmine)** are used for symp
- **Prednisone,** other immunosuppressants (eg, azathio
  mycophenolate mofetil), plasmapheresis, or IVIG ca
  treatment

ung carcinoma; tumor resection may reverse symptoms.
line or guanidine can be given; acetylcholinesterase
ridostigmine) can be added to either regimen.
and azathioprine can be combined or used alone for
sion in cases where a neoplasm cannot be identified and
cause is suspected.

## SIS

er of unclear etiology, but thought to be **T-cell mediated.**
ratio is 3:2, and onset is typically between 20 and 40 years
s more common with increasing distance from the equa-
d. Subtypes are **relapsing-remitting** (most common), 1°
essive, and progressive relapsing.

**ultiple neurologic complaints that are separate in time**
**are not explained by a single lesion.** As the disease pro-
nt deficits can accumulate.
gait unsteadiness, paresthesias, **optic neuritis, ophthal-**
pia, vertigo, nystagmus, urinary retention, sexual and
on, depression, and cognitive impairment are also seen.
cally worsen transiently with hot showers.
edictable but on average occur every 1.5 years, lasting for

ptoms can come and go or be progressive. Those with a
**mitting history** have the **best prognosis.**

ence alone is not sufficient, use MRI and LP as outlined
nostic Criteria for definitive diagnosis.

The clas
MS is sc
ophthal

Pregnan
↓ in MS

patients make a complete or near-complete recovery (ma
The mortality rate is < 5%.

### History/Pe

- Classically presents with progressive (over days), sy
  **paralysis** (distal to proximal), and **areflexia.** In severe
  progress to involve the trunk, diaphragm, and cranial n
- Autonomic and sensory nerves may also be affected,
  **sias** and **autonomic dysregulation.**

### Diagnosis

- Evidence of diffuse demyelination is seen on **nerve c**
  which show ↓ nerve conduction velocity.
- Supported by a **CSF protein level > 55 mg/dL** with lit
  (albuminocytologic dissociation).

### Treatment

- Frequently monitor maximal (negative) inspiratory fo
  capacity to determine whether patient should be ad
  impending **respiratory failure.**
- **Plasmapheresis** and **IVIG** are first-line treatments. Co
  **indicated.**
- Aggressive physical rehabilitation is imperative.

## AMYOTROPHIC LATERAL SCLEROSIS

A **chronic, progressive degenerative disease,** usually
ogy, that is characterized by loss of **upper and lower m**
known as **Lou Gehrig disease** in the United States and **m**
in the United Kingdom. ALS has an unrelenting course
progresses to respiratory failure and death, usually within
sis. Men are more commonly affected than women, an

| | |
|---|---|
| Babinski refl x, pronator drift | Atrophy, fasciculations |

esentation is usually diagnostic. "Bulbar involvement" the tongue [CN XII] or oropharyngeal muscles [CN pathology **above the foramen magnum** and generally ost common differential diagnosis, cervical spondylosis myelopathy, as a cause.

**duction studies** reveal widespread denervation and spon- otentials (fibrillation potentials). Such studies are princi- to exclude other demyelinating motor neuropathies.

cervical spine is done to exclude structural lesions, such dylosis with compressive myelopathy. Especially useful in **ulbar involvement.**

s and patient education. Riluzole may delay disease pro- nate.

## a

ve, global decline in multiple cognitive areas. **Alzheimer r 60–80% of cases.** The differential diagnosis is described **DEMENTIAS.** Take care not to confuse delirium and sychiatry chapter). Table 2.10-10 and the sections below ourse, diagnostic criteria, and treatment of common types

Some 2 onset" A difficult motility, or nasal

Bulbar i **above t** which d spondyl myelopa

SE

| | | |
|---|---|---|
| ual | **Pick bodies** (round intraneuronal inclusions). | MRI/CT show frontotemporal atrophy |
| ual/abrupt | — | CT/MRI reveal ventricular enlargemer |
| pt | Prion proteins on biopsy. | MRI with diffusion weighted imaging FLAIR intensity in the putamen and caudate and is also used to exclude lesions.<br>EEG shows **periodic sharp wave com** |
| ual | Lewy bodies (clumps of α-synuclein proteins). | MRI/CT to rule out other diagnoses. |

- **Early stages:** Physical examination is generally norma status.
- **Late findings:** May include noncognitive neurologi (difficulty with learned motor tasks), and urinary incon

Diagnosis

- **A diagnosis of exclusion** suggested by clinical featur ously progressive **cognitive decline without substan ment.**
- **Definitively diagnosed on autopsy.**
- MRI or CT may show atrophy, especially in temp and can rule out other causes, particularly vascular d

in cognitive functioning.
ted with other symptoms of stroke, such as **sensory** or

ude age, hypertension, diabetes, embolic sources, and a

agnosis of vascular dementia include the presence of
r more of the following:

signs on examination.
hat was **abrupt,** stepwise, or related to stroke.
dicated unless another etiology is suspected.

If a patie
sympto
steady d

evention and treatment of vascular dementia are the same

## DEMENTIA (PICK DISEASE)

form of dementia characterized by **atrophy of the frontal**
.

with **disinhibition** and **significant changes in behavior**
**early in the disease.** Other symptoms include speech dis-
ntiveness, compulsive behaviors, and occasionally extrapy-

dementia rarely begins after age 75.

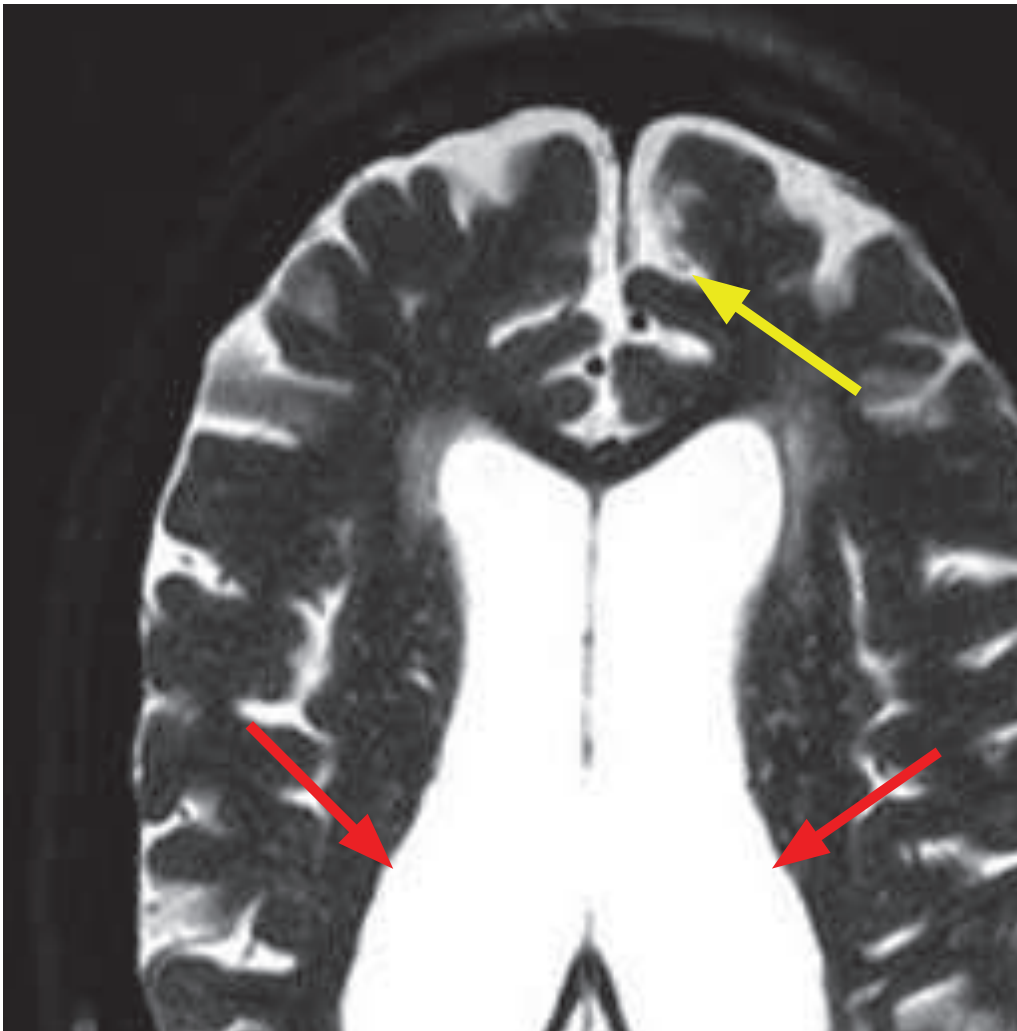
- LP reveals normal CSF pressures and can be therapeu

### Treatment

LP or continuous lumbar CSF drainage for several days r
significant improvement of the patient's symptoms. If so,
peritoneal shunting is the treatment of choice.

## CREUTZFELDT-JAKOB DISEASE

Although it is the most common **prion disease,** CJD re
rare form of dementia. CJD is a member of the transm
**encephalopathies,** all of which are characterized by sp
neuronal loss, and astrocytic proliferation. In CJD, an
resistant prion protein accumulates in the brain.



diagnosis includes limbic encephalitis, Hashimoto (ste-
encephalopathy, and toxic encephalopathy (eg, lithium or

of CSF 14-3-3 and tau protein are seen, indicating rapid
urons.
osis can be made only by brain biopsy or autopsy, but
or detection of periodic sharp wave complexes) may be
ens must be handled with special precautions to prevent

no effective treatment. Most patients die within 1 year of

TIA

r etiology characterized by progressive cognitive changes,
s, and Parkinson-like movement abnormalities such as

**gnitive symptoms** may present around the same time and

dykinesia, rigidity, tremor and shuffling gait with narrow-

rogressive problems with visual-spatial processing, atten-
e dysfunction and memory.
**ptoms** are common and can include hallucinations, delu-
, and anxiety.

oms at
gton

## History/Pe

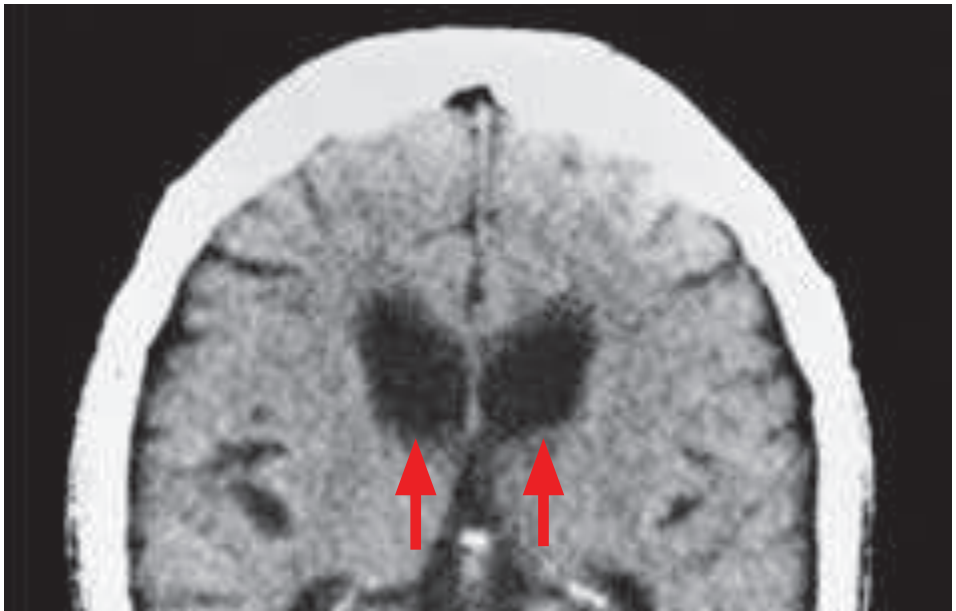
- Presents at 30–50 years of age with gradual onset c
  less, involuntary dancelike movements), **altered beha**
  (begins as irritability, clumsiness, fidgetiness, moodi
  behavior).
- Weight loss and depression may also be seen.

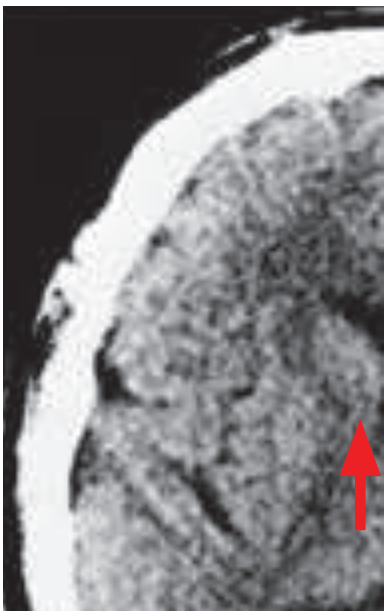
## Diagnosis

- Clinical diagnosis confirmed by genetic testing.
- CT/MRI show cerebral atrophy (especially of the **cau**
  see Figure 2.10-7).

## Treatment

- There is no cure, and disease progression cannot be
  tomatically.
- Reserpine or tetrabenazine can be given to minimiz
  ments. Psychosis should preferably be treated with aty
  to ↓ the risk of extrapyramidal side effects or tardive d
  first-line therapy for depression.
- Genetic counseling should be offered to offspring.





**or** (eg, "pill rolling") with a frequency of **4–6 Hz** that
**h voluntary movement.**
gwheeling."
**:** Slowed movements as well as difficulty initiating move-
nating gait (small, shuffling steps) without arm swing is

**ability:** Stooped posture, impaired righting reflexes, falls.
**tions:** Masked facies, memory loss, and micrographia.

mild antiparkinsonian activity and may improve akinesia,
nor. It can be used for temporary, short-term monotherapy
se of the disease.
Ankle edema and livedo reticularis.
**dopa** combination therapy is the mainstay of treatment.
opamine precursor that can cross the blood-brain barrier.
ks the peripheral conversion of levodopa to prevent the
vodopa (nausea and vomiting).
**fects: Hallucinations,** dizziness, headache, and agitation.
**ects:** Involuntary movements.
**ists** (ropinirole, pramipexole, bromocriptine) can be used
early disease.
Hypotension, somnolence, **confusion, hallucinations,**
**ive gambling.**
MAO-B inhibitor) may be neuroprotective and may ↓ the
a.
**Confusion** and insomnia.
**thyltransferase (COMT) inhibitors** (entacapone or tolca-
ven alone but ↑ the availability of levodopa to the brain
r fluctuations.
**s** (trihexyphenidyl or benztropine)—generally used in
whose primary symptom is tremor.
Dry mouth, blurred vision, constipation, nausea, and uri-



Parkins
feel **TR**

**T**remor (
**R**igidity (
**A**kinesia
**P**ostural



- Park
  defi
- Alzh
  nore
- Mya
  activ

## ° Neoplasms in Adults

| benign vs Malignant | preSentation | |
|---|---|---|
| Diffus —benign<br>Anaplastic—malignant<br>Glioblastoma—malignant | Presentation of astrocytomas depends on location of tumor. Some symptoms include headache, seizures, or focal deficit .<br>Glioblastoma is the most common malignant 1° brain tumor. Progresses rapidly and has a poor prognosis (< 1 year from the time of diagnosis). | Surgical re<br>Radiation<br>have varia |
| Generally benign | Presentation depends on location; often related to cranial neuropathy or is an incidental findin . | Surgical re<br>unresecta |
| Generally benign | Unilateral hearing loss, tinnitus, | Surgical re |





A primitive **neuroectodermal** tumor. Arises from the fourth ventricle or cerebellar vermis and causes ↑ ICP. **Posterior fossa/ infratentorial tumor.**

**Highly malignant but radiosensitive;** may seed the subarachnoid space. May cause obstructive hydrocephalus.

S
ra



The most common **suprasellar** tumor in children. **Calcifi ation** is common.

Benign. May cause hypopituitarism or homonymous hemianopsia.

S

oma reproduced with permission from Hafez RFA. Stereotaxic gamma knife surgery in tr
reliminary result, World J Surg Oncol 2007 Mar 29;5:39. CT of medulloblastoma reproduce
nd Human Services and Armed Forces Institute of Pathology. CT of craniopharyngioma r
J, et al. Craniopharyngioma, Orphanet J Rare Dis. 2007 Apr 10;2:18.

- Hyperventilation (↓ $CO_2$ leads to cerebral vasocon
  ↓ vasogenic edema).
- Corticosteroids (↓ vasogenic edema).
- Mannitol (extraction of free water from brain via os
- Removal of CSF.

- AEDs can be used in patients who have had a seizure.

# Neurocutaneous Disorders

## NEUROFIBROMATOSIS

The most common neurocutaneous disorder. There ar
neurofibromatosis 1 (NF1, or von Recklinghausen syndro
matosis 2 (NF2). Both are **autosomal dominant** diseases.

### History/Pe

- Diagnostic criteria for **NF1** include **two or more** of the
  - Six **café-au-lait spots** (flat, uniformly hyperpigment
  - Two neurofibromas (benign peripheral nerve she
    type (see Figure 2.10-8).
  - **Freckling in the axillary or inguinal area.**
  - **Optic glioma.**
  - Two **Lisch nodules** (pigmented iris hamartomas).
  - Bone abnormality (eg, kyphoscoliosis).
  - A **first-degree relative** with NF1.
- Diagnostic criteria for **NF2** are as follows:
  - **Bilateral vestibular schwannomas** (also known as **a**
  - First-degree relative with NF2 and **either:**
    - Unilateral acoustic neuromas or
    - Two of any of the following tumor types: neuro
      mas, gliomas, or schwannoma



mas
sis
Rx.com.)

and cheeks in the distribution of a butterfly; Figure 2.10-
**een patch** (a rough papule in the lumbosacral region with
onsistency).
are 2° to small benign tumors that grow on the face, eyes,
d other organs. For example:
cardiac rhabdomyoma; renal disease from renal cysts,
, or carcinomas.
tal disability from brain lesions.

are enhanced by a **Wood UV lamp.**

**n:** Evaluate for subependymal giant cell astrocytoma and
ers (potato-like nodules) within the cerebrum in the peri-
rea. If lesions obstruct CSF outflow, obstructive hydro-
develop.
**raphy:** Evaluate for rhabdomyoma of the heart, especially
f the left ventricle (affects > 50% of patients).
**omen:** Evaluate for renal disease (cysts, angiolipoma, and/
).
te for seizure activity.





regressio

Q

A 61-y
the ED
progre
morni
appar
prefer

Q

A 82-y
ED wit
speaki
face a
she sp
chopp

# Aphasia

A general term for speech and language disorders. Usually (eg, strokes, tumors, abscesses) in the "dominant hemisph sphere is dominant in > 95% of right-handed people a handed people.

## BROCA APHASIA

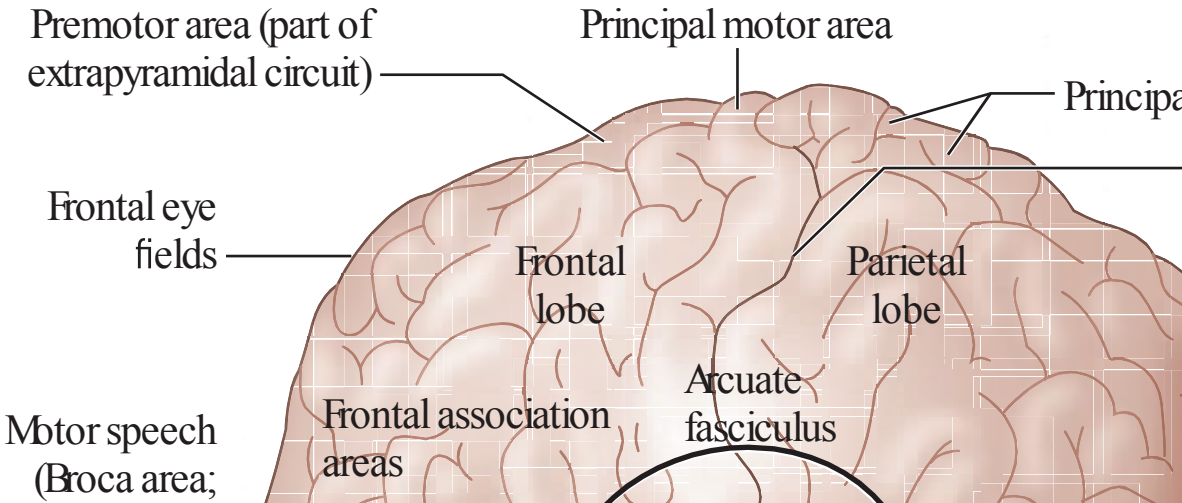
A **disorder of spoken and/or written language productio prehension.** Due to an insult to Broca area in the **poste cortex** (see Figure 2.10-10). Often 2° to a left superior known as **motor aphasia.**

### History/Pe

Presents with **impaired speech production, frustration** deficits, arm and facial **hemiparesis,** hemisensory loss, an muscles. Speech is described as "telegraphic" with few pauses.

### Treatment

**Speech therapy** (varying outcomes with intermediate prog



ding to **"word salad."** Patients are unable to follow com-
ent use of **neologisms** (made-up words) and paraphasic
tions); and show **lack of awareness** of deficits.

**BRO**ca i
**W**ordy.

g etiology and institute speech therapy.

arked by limited to no response to stimuli (ie, a state of
onsiveness). Lesser states of impaired arousal are known
"stupor." Coma is caused by dysfunction of both cere-
the brainstem (pons or higher) due to structural or toxic-
auses include the following:

**ischemic encephalopathy** (eg, postcardiac arrest).
**injury** from high-acceleration trauma (eg, motor vehicle

**n** (eg, cerebral mass lesion, SAH with obstructive hydro-

**ection** (eg, viral encephalitis or advanced bacterial menin-

**em hemorrhage or infarction.**
myelinolysis.
**rbances** (eg, hypoglycemia).
**s** (eg, opiates, benzodiazepines, EtOH, other drugs).
**ure** activity or postictal states.
severe hypothyroidism) or **metabolic dysfunction** (eg, thi-
).

te medical history from witnesses, including current med-

| | | | |
|---|---|---|---|
| dvanced ALS. | | widespread infection, electrolyte disturbances, toxins. | |
| d eyelids. | None. | None. | Nor |
| | Yes. | Yes. | Nor |

cology screen, and blood and CSF cultures. Other
TSH) may be performed based on the clinical index of

- Obtain a **head CT without contrast** before other ima
hemorrhage or structural changes. Imaging should **pr**
the risk of herniation.
- Obtain an MRI to exclude structural changes and ischer
- Rule out catatonia, conversion unresponsiveness, **"loc**
or **persistent vegetative state** (PVS), all of which ca
true coma (see Table 2.10-13).

### Treatment

Initial treatment should consist of the following measures:

- **Stabilize the patient:** Attend to **ABCs.**
- **Reverse the reversible:** Administer **DONT—D**extro
one, and **T**hiamine.
- **Identify and treat the underlying cause** and associated
- **Prevent further damage.**

ysicians
y brain

## Nutritional Deficiencie

| | | |
|---|---|---|
| Korsakoff deme tia | Above plus **anterograde and retrograde amnesia,** horizontal nystagmus, and **confabulations.** | Same as above. Usually occurs in Wernicke syndrome that was treated too late or inadequately. |
| **Peripheral neuropathy;** Combined system disease (CSD) | Gradual, progressive onset. Symmetric paresthesias, stocking-glove sensory neuropathy, leg stiffnes , spasticity, paraplegia, bowel and bladder dysfunction, sore tongue, and dementia. Associated with elevated methylmalonic acid levels. | Patients with pernicious anemia; strict vegetarians; status post gastric or ileal resection; ileal disease (eg Crohn); alcoholics or other with malnutrition. |
| olate deficien y | Irritability; personality changes without the neurologic symptoms of CSD. | Alcoholics. |

ocysteine and an ↑ risk of vascular events.

s humor produced by the ciliary body on the iris travels

Closed

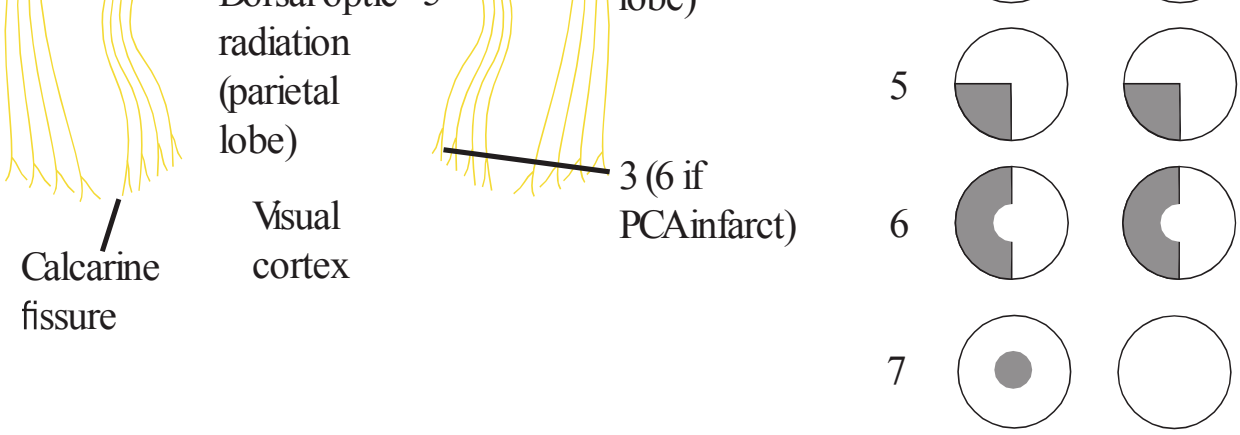



**FIGURE 2.10-11. Visual field defects (1)** Right anopsia. **(2)** Biter
(pituitary lesion, chiasm). **(3)** Left homonymous hemianopsia. **(4)** Left
anopsia (right temporal lesion, MCA). **(5)** Left lower quadrantic anopsi
MCA). **(6)** Left hemianopsia with macular sparing (PCA infarct). **(7)** C
lar degeneration). (Reproduced with permission from Le T et al. First Aid for the USMLESt
McGraw-Hill; 2015.)

## CATARACTS

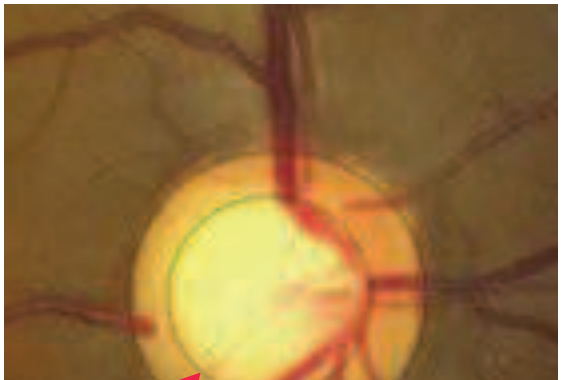
Lens opacification resulting in obstructed passage of lig
diabetes, hypertension, advanced age, and exposure to rad

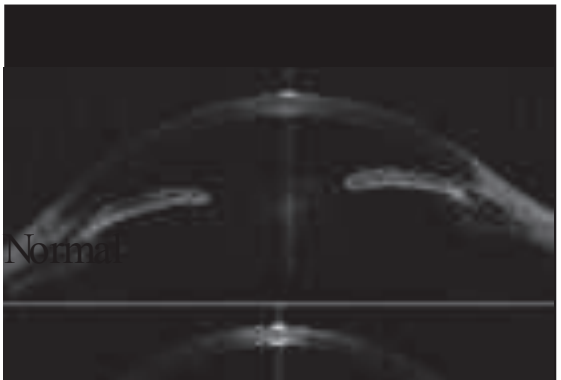
### History/Pe

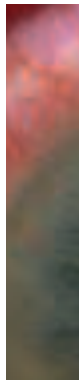
Presents with loss of visual acuity and difficulty with night

### Treatment

Surgical lens removal and replacement.





| | |
|---|---|
| eme, sudden-onset eye pain, blurred vision, headache, ausea, and vomiting.<br>rd, red eye is seen; the pupil is dilated and nonreactive light. | Should be suspected in pati need **frequent lens chan** visual disturbances, and i usually asymptomatic un<br>Characterized by gradual lo<br>**Cupping** of the optic nerve exam (see Figure 2.10-12) |
| gnosis is based on clinical history and examination.<br>d pupil and hard, red eye. | Tonometry, ophthalmoscop nerve and cupping of optic most important. |
| **edical emergency** that can cause blindness. Treatment ↓ IOP is as follows:<br>drops (timolol, pilocarpine).<br>emic medications (oral or IV acetazolamide, IV annitol).<br>**er peripheral iridotomy,** which creates a hole in e peripheral iris, is **curative** and can be performed ophylactically.<br>ot give any medications that cause pupillary dilation. | Treat with topical β-blocker ↓ aqueous humor produc<br>Pilocarpine to ↑ aqueous ou<br>Carbonic anhydrase inhibito used.<br>If medication fails, laser trab can improve aqueous dra |

## ULAR DEGENERATION

ng whites, women, smokers, and those with a family history.



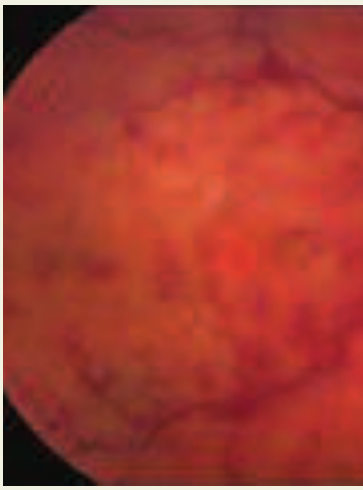
**inless loss of central vision.** Early signs include distortion

- Photodynamic therapy using a laser to selectively
  for coagulation. May be useful in conjunction with

## RETINAL VASCULAR OCCLUSION

Occurs in **elderly patients** and is often idiopathic (see Tab

**inal Artery vs Central Retinal Vein Occlusion**

| Central Retinal Artery Occlusion | Central Retinal Vein |
|---|---|
|  |  |
| th sudden, **painless, unilateral blindness.** The uggishly reactive to direct light. sent with a **cherry-red spot** on the fovea w), retinal swelling (whitish appearance to the er 1 yer), and retinal arteries that may appear . | Presents with rapid, **painless** vision severity. Associated with hyperte A swollen optic disk with hemorrha retinal hemorrhages, cotton-woo edema may be seen on fundusco |
| ge; intra-arterial thrombolysis within 8 hours. | Laser photocoagulation (variable res |

nage reproduced with permission from USMLE-Rx.com. Central retinal vein occlusion im
ser ME. Intravitreal bevacizumab in the treatment of neovascular glaucoma secondary t

Pregnancy 296
Y 296
296
PREGNANCY 296
297
sting 299
300
PLASMA PROTEIN A 300
NG 300
301
302
ns 302
302
f Pregnancy 305
very 306
306
To RING 306
EILLANCE 307
ANESTHESIA 311
s of Pregnancy 311
I 311
312
c HYPERTENSION 313
PSIA 314
UTI AND PYELONEPHRITIS DURING PREG
ANTEPARTUM HEMORRHAGE
Obstetric Complications of Pregn
ECTOPIC PREGNANCY
INTRAUTERINE GROWTH RESTRICTION
FETAL MACROSOMIA
POLYHYDRAMNIOS
OLIGOHYDRAMNIOS
RH ISOIMMUNIZATION
GESTATIONAL TROPHOBLASTIC DISEASE
MULTIPLE GESTATIONS
Abnormal Labor and Delivery
SHOULDER DYSTOCIA
FAILURE TO PROGRESS
RUPTURE OF MEMBRANES
PRETERM LABOR
FETAL MALPRESENTATION
INDICATIONS FOR CESAREAN SECTION
EPISIOTOMY
Puerperium
POSTPARTUM HEMORRHAGE
POSTPARTUM INFECTIONS
SHEEHAN SYNDROME (POSTPARTUM PITU
LACTATION AND BREASTFEEDING
MASTITIS

tional age or an infant weighing > 500 g.
    - In prenatal assessment, P expresses the number of number of preterm deliveries, the number of abor ber of living children (where P stands for "para").
- **Developmental age (DA):** The number of weeks and tion; usually unknown.
- **Gestational age (GA):** The number of weeks and days first day of the last menstrual period (LMP). GA can by:
    - **Fundal height:** Umbilicus – 20 weeks + 2–3 cm/we
    - **Fetal heart tones (Doppler):** Typically 10–12 week
    - **Quickening, or appreciation of fetal movemen** weeks at the earliest.
    - **Ultrasonography:**
        - Measures fetal crown-rump length (CRL) at 6–1
        - Measures biparietal diameter (BPD), femur abdominal circumference (AC) from 13 weeks.
        - Ultrasound measurement of GA is most reliable mester.

## DIAGNOSIS OF PREGNANCY

### β-hCG

- The standard for diagnosing pregnancy.
- Produced by the placenta; peaks at 100,000 mIU/mL b
- ↓ throughout the second trimester; levels off in the thi
- **hCG levels double approximately every 48 hours nancy.** This is often used to diagnose ectopic pregnan abnormal.

### Ultrasonography

- Used to confirm an intrauterine pregnancy.

**nal and uterine/body weight changes in normal pregnancy.** (Reproduced with permissio
n ed. New York, NY: McGraw-Hill; 2007.)

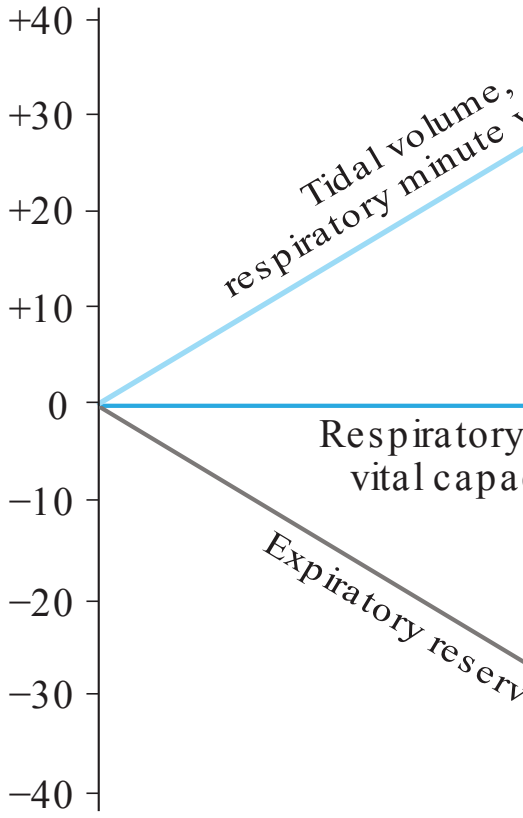
# Care

l care is to prevent, diagnose, and treat conditions that can
omes in pregnancy. Expected weight gain, nutrition, and
dations are outlined in Table 2.11-1. See Table 2.11-2 for
ors that can cross the placenta.

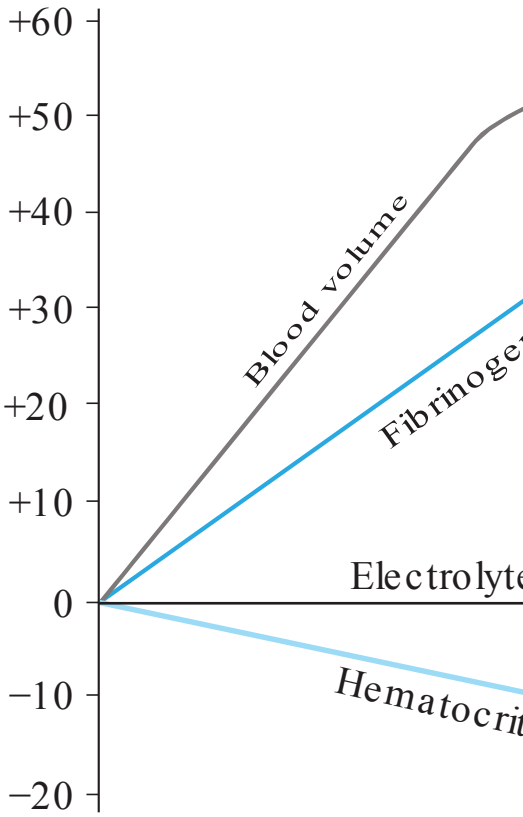
**ommendations for Standard Prenatal Care**

| r eco MMen dat io n S |
|---|
| elines for weight gain in pregnancy:<br>**cessive gain:** > 1.5 kg/month.<br>**adequate gain:** < 1.0 kg/month.<br>elines according to prepregnancy BMI:<br>**derweight** (BMI < 19.8): 12–18 kg.<br>**ceptable** (BMI 19.8–26.0): 11–16 kg.<br>**erweight** (BMI 26.1–29.0): 7–11 kg.<br>**verely overweight** (BMI > 29.0): 5–9 kg. |
| elines for nutritional supplementation:<br>additional 100–300 kcal/day; 500 kcal/day during breastfeeding.<br>**lic acid supplements** (↓ neural tube defects for **all** reproductive-age<br>men): 0.4 mg/day, or 4 mg/day for women with a history of neural tube<br>fects in prior pregnancies. |

| | |
|---|---|
| distention | |
| Peripheral vascular resistance | Progressive decrease to term. |
| Respiratory rate | Unchanged. |
| Tidal volume | Increases by 30–40%. |
| Expiratory reserve | Gradual decrease. |
| Vital capacity | Unchanged. |
| Respiratory minute volume | Increases by 40%. |




| | |
|---|---|
| Volume | Increases by 50% in second trimester. |
| Hematocrit | Decreases slightly. |
| Fibrinogen | Increases. |
| Electrolytes | Unchanged. |




| | |
|---|---|
| Sphincter tone | Decreases. |



Treponema pallidum
Listeria monocytogenes
Parvovirus B19

# Diagnostic Testing

es a typical prenatal diagnostic testing schedule by week. llow describe each recommended screening modality.

**natal Diagnostic Testing Schedule**

| prenatal diagnostic testing |
|---|
| **eks 0–28:** Every 4 weeks.<br>**eks 29–35:** Every 2 weeks.<br>**eks 36–birth:** Every week. |
| **me:** CBC, Rh factor, type and screen.<br>**ectious disease:** UA and culture, rubella antibody titer, HBsAg, RPR/RL, cervical gonorrhea and chlamydia, PPD, HIV, Pap smear (to check for plasia). Consider HCV and varicella based on history.<br>**ndicated:** Hb $A_{1c}$, sickle cell screening.<br>**cuss genetic screening:** Tay-Sachs disease, cystic fib osis. |
| er PAPP-A + nuchal transparency + free β-hCG ± CVS. |
| er maternal serum α-fetoprotein (MSAFP) or quad screen (AFP, estriol, CG, and inhibin A) ± amniocentesis. |

- Measurement results depend on accurate gestationa
- MSAFP is rarely tested alone, as quad screening detecting chromosomal abnormalities.

- **Elevated MSAFP (> 2.5 MoMs) is associated with:**
  - Open–neural tube defects (anencephaly, spina bifi
  - Abdominal wall defects (gastroschisis, omphalocele
  - Multiple gestation.
  - Incorrect gestational dating.
  - Fetal death.
  - Placental abnormalities (eg, placental abruption).
- **Reduced MSAFP (< 0.5 MoM) is associated with:**
  - Trisomy 21 and 18.
  - Fetal demise.
  - Incorrect gestational dating.

## PREGNANCY-ASSOCIATED PLASMA PROTEIN A

- Recommended at weeks 9–14.
- PAPP-A + nuchal transparency + free β-hCG can dete Down syndrome and ~ 95% of cases of trisomy 18.
- Advantages:
  - A screen of pregnant women (> 35 years of age).
  - Available earlier than CVS and less invasive than C

## CHORIONIC VILLUS SAMPLING

Table 2.11-5 outlines the relative advantages and disadva amniocentesis (see also Figure 2.11-3).

**TABLE 2.11-4. Quad Screening**

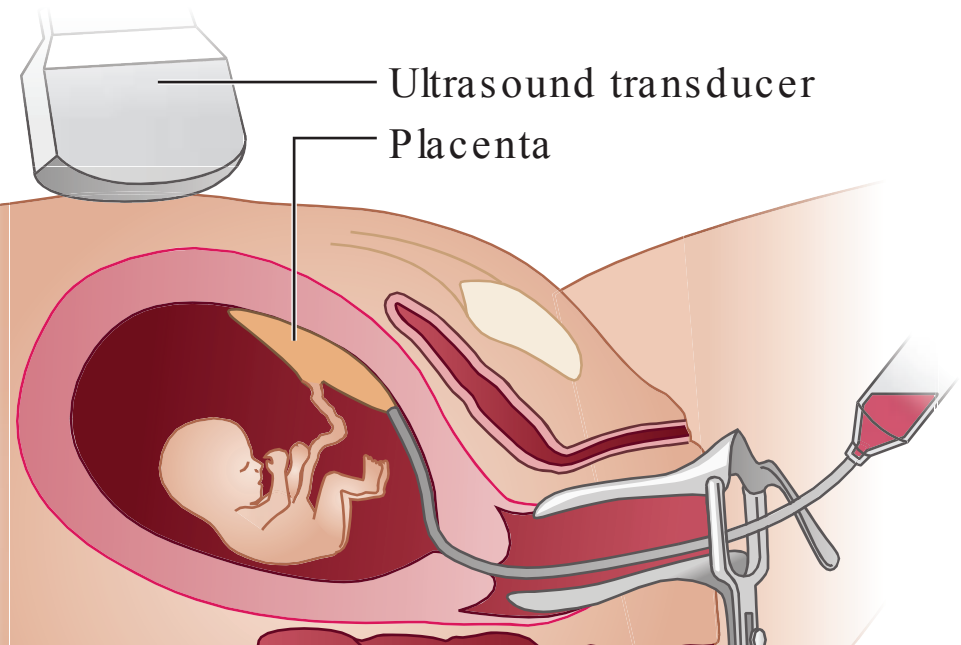
ilable at an earlier GA.

| | | |
|---|---|---|
| k of fetal loss is relatively high (1%).<br>nnot detect open–neural tube efects.<br>b defects are associated with CVS t < 9 weeks. | PROM, chorioamnionitis, fetal-maternal hemorrhage. | May b<br>conce<br>circula |

lowing:

vill be > 35 years of age at the time of delivery.
with an abnormal quad screen.
pregnancy to obtain fetal blood type or to detect fetal

lung maturity via a lecithin-to-sphingomyelin ratio ≥ 2.5
presence of phosphatidylglycerol (performed during the



sequelae include the following:

- Premature delivery.
- CNS abnormalities.
- Anemia.
- Jaundice.
- Hepatosplenomegaly.
- Growth retardation.

- The most common pathogens involved can be remem of the **TORCHeS** mnemonic (see also Table 2.11-7).

## Spontaneous Abortion

The loss of products of conception (POC) prior to the nancy. More than 80% of cases occur in the first trimeste follows:

- **Chromosomal abnormalities:** A factor in approximat the first trimester, 20–30% in second-trimester losses, trimester losses.
- **Maternal factors:**
  - **Inherited thrombophilias:** Factor V Leiden, proth bin, proteins C and S, methylene tetrahydrofolate mocysteinemia).
  - **Immunologic issues:** Antiphospholipid antibodie tors.
  - **Anatomic issues:** Uterine and cervical abnorma cervix, cervical conization or loop electrosurgical (LEEP), cervical injury, DES exposure.
  - **Endocrinologic issues:** Diabetes mellitus (DM), h gesterone deficiency.
  - **Other:** Maternal trauma, ↑ maternal age, infecti cies.

| | |
|---|---|
| | Virilization of female fetuses; advanced genital development in male fe |
| | Neural tube defects, finge nail hypoplasia, microcephaly, developmenta |
| | Bowel atresias; congenital malformations of the heart, limbs, face, and G cerebral infarctions. |
| | Clear cell adenocarcinoma of the vagina or cervix, vaginal adenosis, abr uterus or testes, possible infertility. |
| | ↑ SAB rate; stillbirths. |
| | Congenital heart disease (Ebstein anomaly). |
| | ↑ SAB rate. |
| | Cerebral atrophy, microcephaly, mental retardation, spasticity, seizures, |
| | IUGR, mental retardation, microcephaly, dysmorphic craniofacial feature hypoplasia. |
| | Microcephaly, mental retardation. Medical diagnostic radiation deliveri teratogenic risk. |
| amycin | Hearing loss; CN VIII damage. |
| | Permanent yellow-brown discoloration of deciduous teeth; hypoplasia |
| | Bilateral limb deficiencie , anotia and microtia, cardiac and GI anomalie |
| | Cleft lip or cleft palate, cardiac defects, microcephaly, mental retardatio |

| | | | |
|---|---|---|---|
| ntal | Purpuric "blueberry muf n" rash.<br>Cataracts.<br>Mental retardation.<br>Hearing loss.<br>PDA. | Serologic testing. | Symptomatic. |
| tal. | Petechial rash.<br>Periventricular calcific tions. | Urine culture; PCR of amniotic flui . | Postpartum ganciclovir. |
| n if has ns; tal n is | Skin, eye, and mouth infections.<br>Life-threatening CNS/systemic infection. | Serologic testing. | Acyclovir. |
| nilk. | Often asymptomatic.<br>Failure to thrive.<br>Bacterial infections.<br>↑ incidence of upper and lower respiratory diseases. | ELISA, Western blot. | HAART. |
| n; tal n is | Maculopapular skin rash.<br>Lymphadenopathy.<br>Hepatomegaly.<br>"Snuffl ": mucopurulent rhinitis. | Dark-field microscopy, VDRL/ RPR, FTA-ABS. | Penicillin. |

| | | |
|---|---|---|
| **rtial POC expulsion;** bleeding/ mild cramping. sible tissue on exam. | **Open os.** POC present on ultrasonography. | Manu if < 12 use m mana SABs. |
| erine bleeding and cramps. **No POC pulsion.** | **Open os** ± ROM. POC present on ultrasonography. | |
| **amping, loss of early pregnancy mptoms.** No bleeding. | **Closed os.** No fetal cardiac activity; POC present on ultrasonography. | |
| ul-smelling discharge, abdominal pain, fever, and cervical motion tenderness; ± POC expulsion. aternal mortality is 10–15%. | Hypotension, hypothermia, ↑ WBC count. Blood cultures. | MUA c |
| sence of fetal cardiac activity > 20 eeks GA. | Uterus small for GA; no fetal heart tones or movement on ultrasonography. | Induc (D&E) weeks |

**nancy:** Gestational sac > 25 mm without a fetal pole or cardiac activity when CRL > 7 mm on transvaginal ultra-

tation, speculum exam, ultrasonography and serum

can identify:
al sac 5–6 weeks from the LMP

| | |
|---|---|
| | (high dose), repeated up to 3 times.<br>**Surgical management:**<br>▪ Manual uterine aspiration (MUA).<br>▪ D&C with vacuum aspiration. |
| Second<br>(10% TABs) | **Obstetric management:** Induction of labor (typically with prostaglandins, amniotomy, and oxytocin).<br>**Surgical management:** D&E. |

# Normal Labor and Delivery

## OBSTETRIC EXAMINATION

- Leopold maneuvers are used to determine fetal lie (lo verse) and, if possible, fetal presentation (breech or cep
- **Cervical examination:**
  - Evaluate dilation, effacement, station, cervical po consistency.
  - Confirm or determine fetal presentation.
  - Determine fetal position through palpation of the f tanelles.
  - **Conduct a sterile speculum exam if ROM is susp**
  - Determine station, or engagement of the fetal he through the ischial spines of the maternal pelvis. ⊖ superior to this line; ⊕ station = fetal head inferior
- Table 2.11-10 depicts the normal stages of labor.

## FETAL HEART RATE MONITORING

| | | | |
|---|---|---|---|
| e cervical dilation<br>y of infant. | 0.5–3.0 hours | 5–30 minutes | Neonate go<br>movement |
| of infant to<br>f placenta. | 0–0.5 hour | 0–0.5 hour | Uterus con<br>establish h |

**mplications:** Review FHR tracings.
**labor:** Every 15 minutes.
**of labor:** Every 5 minutes.

## REvaluation

110–160 bpm):
**bpm:** Bradycardia. Can be caused by congenital heart
s or by severe hypoxia (2° to uterine hyperstimulation,
, or rapid fetal descent).
**bpm:** Tachycardia. Causes include hypoxia, maternal
al anemia.
Figures 2.11-4 and 2.11-5.
**bility:** Indicates severe fetal distress.
**iability:** < 6 bpm. Indicates fetal hypoxia or the effects of
nesium, or sleep cycle.
**bility:** 6–25 bpm.
**ability:** > 25 bpm. May indicate fetal hypoxia; may occur
variability.
**ariability:** Points to serious fetal anemia; a pseudosinusoi-
ay also occur during maternal meperidine use.
Onset of an ↑ in FHR > 15 beats above baseline to a peak
Reassuring because they indicate fetal ability to appropri-
the environment.
See Table 2.11-11.

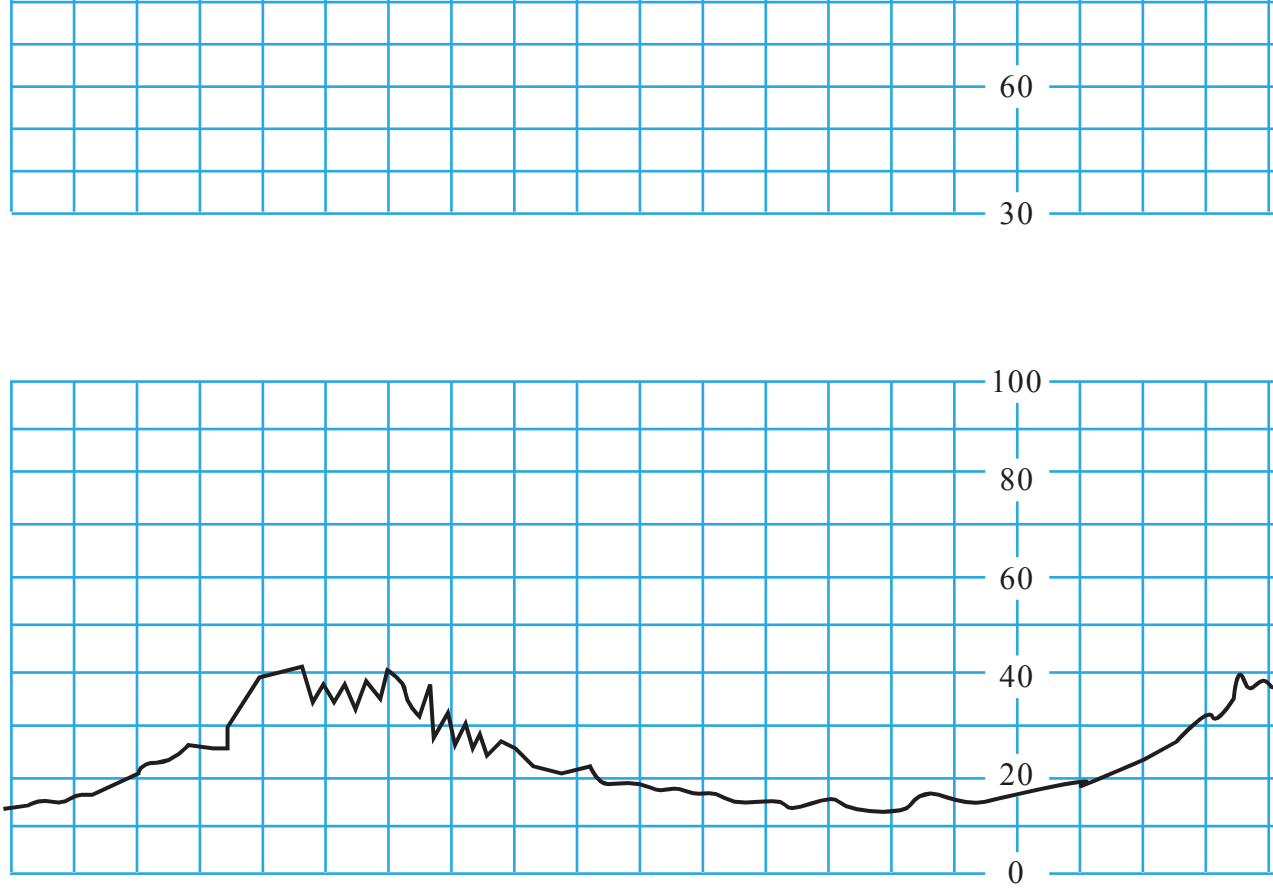
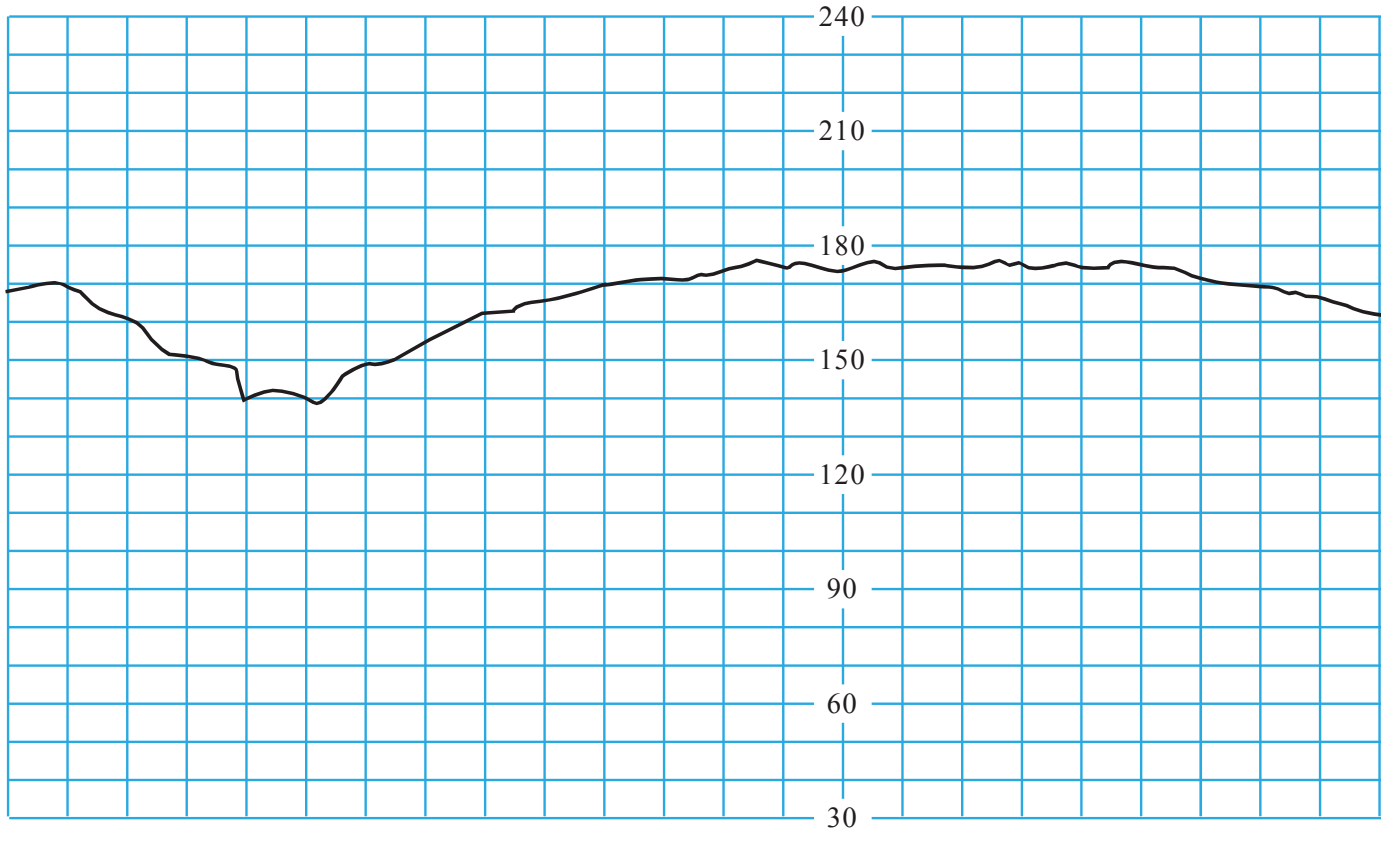
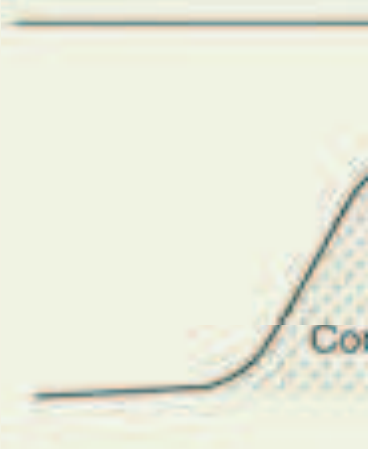



**FIGURE 2.11-4. Varying (variable) fetal heart rate decelerations**






| | | |
|---|---|---|
| apparent, gradual (onset > 30 seconds) ↓ in FHR n to baseline whose dir, and recovery occur beginning, peak, and end contraction, respectively. | Uteroplacental insufficie y and fetal hypoxemia. |  |
| (onset to nadir in < 30 visually apparent ↓ in m below baseline lasting nds but < 2 minutes. | Umbilical cord compression. |  |

ssion from Cunningham FC et al. Williams Obstetrics, 23rd ed. New York, NY: McGraw-Hill,

**NST):**

ith the mother resting in the lateral tilt position (to pre

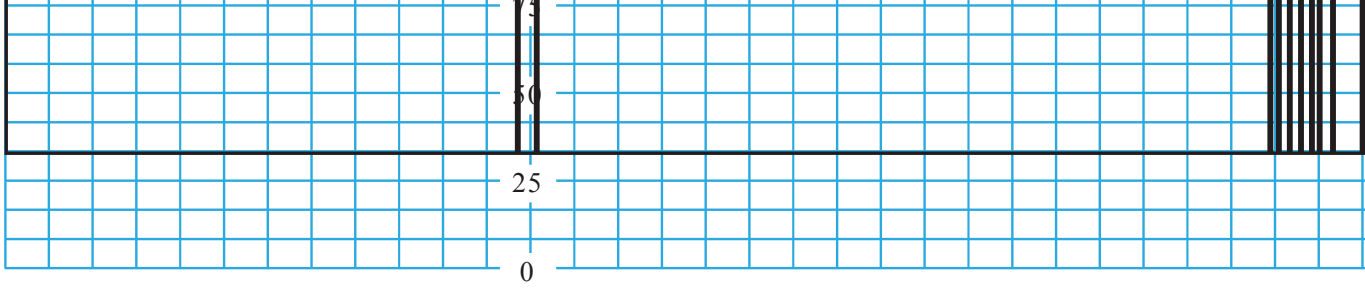



**FIGURE 2.11-6. Reactive nonstress test.** (Adapted with permission from Obstetrics, 23rd ed. New York, NY: McGraw-Hill; 2010.)

- **Contraction stress test (CST):**
  - Performed in the lateral recumbent position.
  - FHR is monitored during spontaneous or induced tion or oxytocin) contractions.
  - Reactivity is determined from fetal heart monitorin
  - The procedure is contraindicated in women with rupture or known placenta previa; those with a hi gery; and those who are at high risk for preterm labo
  - **"Positive" CST:**
    - Defined by late decelerations following 50% tions in a 10-minute window.
    - Raises concerns about fetal compromise.
    - Delivery is usually warranted.
  - **"Negative" CST:**
    - Defined as no late or significant variable dece minutes and at least three contractions.
    - Highly predictive of fetal well-being in conjun NST.
  - **"Equivocal" CST:** Defined by intermittent late nificant variable decelerations.
- **Biophysical Profile (BPP):** Uses real-time ultrasono score of 2 (normal) or 0 (abnormal) to five parameter ing, movement, amniotic fluid volume, and NST. Sco
  - **8–10:** Reassuring for fetal well-being.

pudendal nerve, S2–S4).
of a medical contraindication, maternal request is a suffi-
dication for pain relief during labor.
indications to regional anesthesia (epidural, spinal, or
clude the following:
aternal hypotension.
gulopathy.
e of a once-daily dose of low-molecular-weight heparin
urs.
aternal bacteremia.
n over the site of needle placement.
d by a mass lesion.

# Complications of Pregnancy

## MIDARUM

not related to other causes, **acute starvation** (usually large
ght loss (usually at least a 5% ↓ from prepregnancy weight).

in first pregnancies, multiple gestations, and molar preg-

estradiol have been implicated in its pathophysiology.

orning sickness" (which usually starts between weeks 4 and
resolves prior to 16 weeks, but can occur throughout preg-
gastroenteritis, hyperthyroidism, and neurologic conditions.

evel and perform ultrasonography to rule out molar preg-

If "morn
the first
gravidar

The first
hyperem
**molar p**
β-hCG.

erance or impaired fasting glucose, or HDL-C < 35 m
> 250 mg/dL.

## Gestational Diabetes Mellitus

Carbohydrate intolerance of variable severity that is firs
pregnancy. Occurs in 3–5% of all pregnancies, usually di
trimester (24–28 weeks).

### History/Pe

- Typically asymptomatic.
- May present with edema, polyhydramnios, or a large-fo
  percentile).

### Diagnosis

- **Screen with a 1-hour 50-g glucose challenge test:**
  - Venous plasma glucose is measured 1 hour later.
  - **Performed at 24–28 weeks.**
  - Values ≥ 140 mg/dL are considered abnormal.
- **Confirm with an oral 3-hour (100-g) glucose toleran**
  ing any two of the following:
  - **Fasting:** > 95 mg/dL.
  - **1 hour:** > 180 mg/dL.
  - **2 hours:** > 155 mg/dL.
  - **3 hours:** > 140 mg/dL.

### Treatment

- **Mother:**
  - Start with the **ADA diet,** regular exercise, and strict
    (four times a day).
  - Tight maternal glucose control (fasting ≤ 95 mg/d
    dial ≤ 140 mg/dL and 2-hour postprandial ≤ 120 m
    comes.

tational
sulin if
fetal
t 34

almologic, neural tube, and cardiac evaluation to assess
damage.
control (diet, exercise, insulin therapy, and frequent self-
o minimize fetal defects. **Fasting morning:** ≤ 95 mg/dL.
stprandial: < 140 mg/dL.
**stprandial:** < 120 mg/dL.
glucose 60–99 mg/dL.

:
en for developmental anomalies (16–18 weeks).
graphy to determine fetal age and growth (18–20 weeks).
o to evaluate for cardiac anomalies (22–24 weeks).
:
l surveillance (eg, NST, CST, BPP). (If poor glucose con-
all vessel disease, start at 26 weeks).
maternal DM has been poorly controlled or fetal param-
concern.
asonograms for fetal growth.
**postpartum:**
normoglycemia (80–100 mg/dL) during labor with an IV
ip and hourly glucose measurements.
early delivery in the setting of poor maternal glucose con-
lampsia, macrosomia, or evidence of fetal lung maturity.
delivery should be considered in the setting of an esti-
l weight (EFW) > 4500 g.
e breastfeeding with an appropriate ↑ in caloric intake.

If UA be
think pr

Hypergl
suggest
should b
diabetes

| | |
|---|---|
| Polyhydramnios. | RDS. |
| Postpartum hemorrhage. | Birth injury (eg, shou |
| Maternal mortality. | Perinatal mortality. |

### Treatment

- Monitor BP closely.
- Treat with appropriate antihypertensives (eg, methyld dipine).
- **Do not give angiotensin-converting enzyme inhi diuretics.**
  - ACEIs are known to lead to uterine ischemia.
  - Diuretics can aggravate low plasma volume to the p emia.

### Complications

Similar to those of preeclampsia (see below).

## PREECLAMPSIA AND ECLAMPSIA

Distinguished as follows:

- **Preeclampsia:**
  - New-onset hypertension (systolic BP ≥ 140 mm Hg mm Hg) **and**
  - Proteinuria (> 300 mg of protein in a 24-hour p > 20 weeks GA.
- **Eclampsia:** New-onset grand mal seizures in women w
- **HELLP syndrome:** A variant of preeclampsia with a p
  - Consists of hemolytic anemia, elevated liver enzy lets (see mnemonic).

oliguria (< 500 mL/24 hours).

**Cerebral changes: Headache,** somnolence.

**Visual changes: Blurred vision,** scotomata.

**Other:** Hyperactive refl xes/clonus; **RUQ pain;** hemolysis, elevated liver enzymes, thrombocytopenia (HELLP syndrome).

The most common signs preceding an eclamptic attack are **headache, visual changes,** and **RUQ/epigastric pain.**

Seizures are severe if not controlled with anticonvulsant therapy.

**reeclampsia/eclampsia is delivery of the fetus.**

**m or worsening preeclampsia:** Induce delivery with IV staglandin, or amniotomy.

**m:** Treat with modified bed rest and expectant manage-

**apartum seizures with a continuous magnesium sulfate**

r signs of magnesium toxicity (loss of DTRs, respiratory coma).

seizure prophylaxis for 24 hours postpartum.

gnesium toxicity with IV calcium gluconate.

**psia:**

with labetalol and/or hydralazine (goal < 160/110 mm Hg ic BP of 90–100 mm Hg to maintain fetal blood flow).

**magnesium sulfate drip.**

duction or C-section when the mother is stable.

Signs of
persiste
visual di
pain, an

30–40% will subsequently develop UTI or pyelonephrit
sistent untreated bacteriuria places patient at a higher ris
low birth weight, and perinatal mortality. Escherichia col
70–90% of infections.

### History/Pe

- **Asymptomatic bacteriuria:** ⊕ urine culture on first-tri
  colonies).
- **UTI:** Dysuria, urinary urgency and frequency.
- **Pyelonephritis:** Same as UTI, + fever and costovertebr

### Diagnosis

⊕ urine culture.

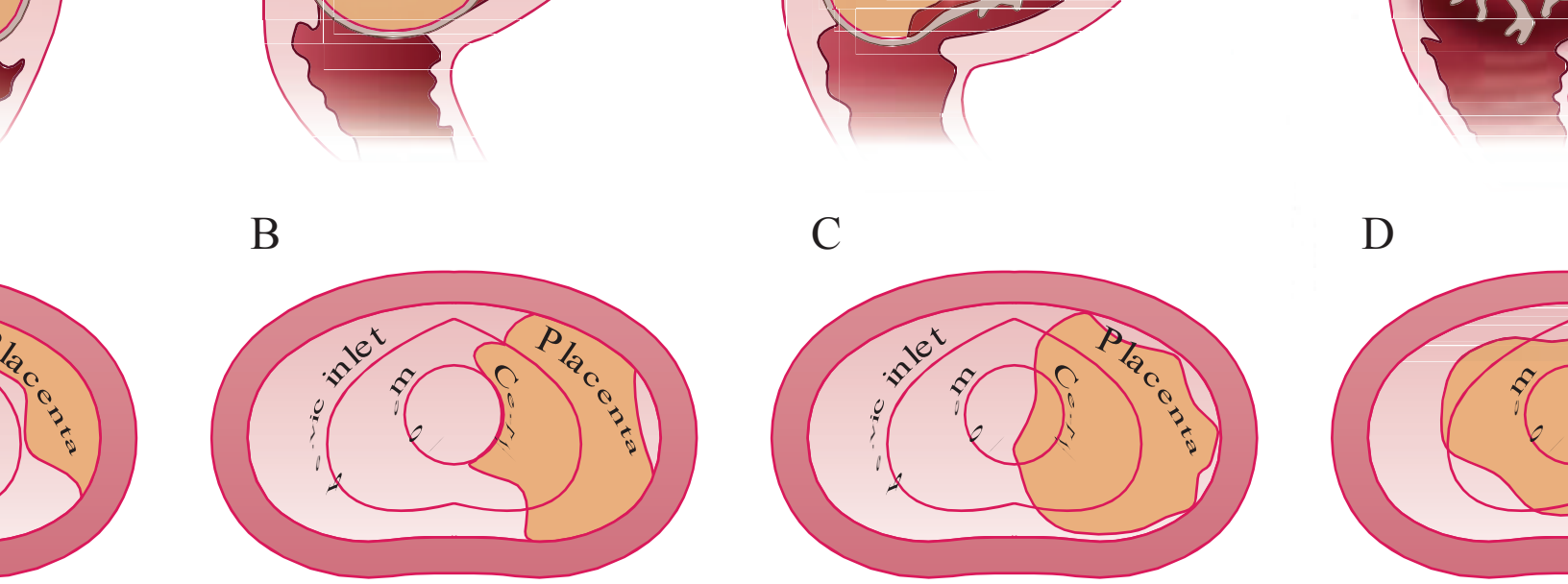
### Treatment

- **Asymptomatic bacteriuria and UTI:** 3–7 days nitrofu
  or amoxicillin-clavulanate. Follow-up culture at 1 weel
- **Pyelonephritis:** Admit to hospital, IV fluids, IV third-
  sporins; Suppressive antibiotics with agent culture sus
  der of pregnancy and follow-up culture.

## ANTEPARTUM HEMORRHAGE

- Any bleeding that occurs after 20 weeks' gestation.
- Complicates 3–5% of pregnancies.
- The most common causes are **placental abruption** a
  (see Table 2.11-14 and Figure 2.11-7).
- Other causes include other forms of abnormal placen
  accreta), ruptured uterus, genital tract lesions, and trau

| | | |
|---|---|---|
| 1 in 100. | 1 in 200. | 1 in 2 |
| Hypertension, abdominal/pelvic trauma, tobacco or cocaine use, previous abruption, rapid decompression of an overdistended uterus, excessive stimulation. | Prior C-sections, grand multiparity, advanced maternal age, multiple gestation, prior placenta previa. | Multi place arter place |
| **Painful, dark** vaginal bleeding that does not spontaneously cease.<br>Abdominal pain; uterine hypertonicity.<br>Fetal distress. | **Painless, bright red** bleeding that often ceases in 1–2 hours with or without uterine contractions.<br>Usually no fetal distress. | **Pain mem** |
| Primarily clinical.<br>Transabdominal/transvaginal ultrasonography sensitivity is only 50%; look for a retroplacental clot. Most useful for ruling out previa. | Transabdominal/transvaginal ultrasonography sensitivity is > 95%; look for an abnormally positioned placenta. | Trans colon passi |
| Stabilize patients with mild abruption and a premature fetus; **manage expectantly** (hospitalize; start IV and fetal monitoring; type and cross-match blood; bed rest).<br>**Moderate to severe abruption: Immediate delivery** is indicated (vaginal delivery with amniotomy | **Do not perform a vaginal exam!**<br>Stabilize patients with a premature fetus; manage expectantly. Give tocolytics.<br>Serial ultrasonograms to assess fetal growth; resolution of partial previa.<br>Betamethasone 28–32 weeks to help with fetal lung maturity. | **Acut C-s**<br>**Diag Ste he ho for sch** |




**mplantation. (A)** Normal placenta. **(B)** Low implantation. **(C)** Partial placenta previ

from DeCherney AH. Current Obstetric &Gynecologic Diagnosis &Treatment, 8th ed. Stamford, CT: Appleton &L

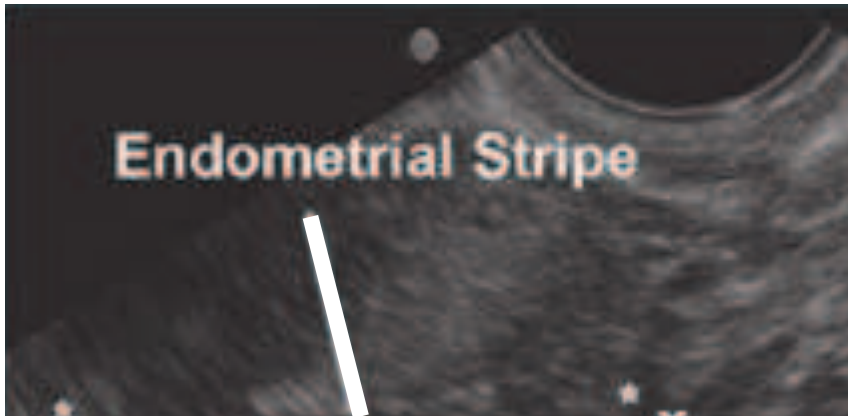
r

## Diagnosis

Approach a woman of reproductive age presenting with a ruptured ectopic pregnancy until proven otherwise.

- Look for a ⊕ pregnancy test and a transvaginal ultraso empty uterus (see Figure 2.11-8).
- Confirm with a serial β-hCG without appropriate β-hC





ne 10th percentile for GA.

the following:

ic disease leading to uteroplacental insufficiency (intra-
, hypertension, anemia).
nce abuse.

ons.

undal height measurements with ultrasonography.
ography for EFW.

erlying etiology, and correct if possible.
**near due date,** administer steroids (eg, betamethasone) to
ung maturity; requires 48 hours prior to delivery.
onitoring with NST, CST, BPP, and umbilical artery Dop-

status near term may prompt delivery.

y and mortality.

A

th weight > 95th percentile. A common sequela of gesta-

- Twin-twin transfusion syndrome.
- **Hx/PE:** Usually asymptomatic.
- **Dx:** Fundal height greater than expected. Evaluation raphy for fetal anomalies, glucose testing for DM, and
- **Tx:** Etiology specific.
- **Complications:** Preterm labor, fetal malpresentation,

## OLIGOHYDRAMNIOS

- An AFI < 5 on ultrasonography. Usually asymptomatic distress may be present.
- Etiologies include the following:
  - Fetal urinary tract abnormalities (eg, renal agenesis
  - Chronic uteroplacental insufficiency.
  - ROM.
- **Dx:** The sum of the deepest amniotic fluid pocket in quadrants on ultrasonography.
- **Tx:** Rule out inaccurate gestational dates. Treat the possible.
- **Complications:**
  - Associated with a 40-fold ↑ in perinatal mortality.
  - Other complications include musculoskeletal abn foot, facial distortion), pulmonary hypoplasia, umb sion, and IUGR.

## RH ISOIMMUNIZATION

In this condition, fetal RBCs leak into the maternal circu anti-Rh IgG antibodies form that can cross the placenta, l of fetal Rh RBCs (**erythroblastosis fetalis;** see Figure 2.1

Rh ⊖ at 28 weeks and the father is Rh ⊕ or unknown,
Rh immune globulin).
⊕, give the mother RhoGAM postpartum.
to Rh-⊖ mothers who undergo abortion or who have had
ancy, amniocentesis, vaginal bleeding, or placenta previa/
ion. **Type and screen is critical;** follow β-hCG closely
nancy for 1 year.

hen fetal hemoglobin is < 7 g/dL.
d acidosis, kernicterus, prematurity, death.

## PHOBLASTIC DISEASE

ative trophoblastic abnormalities that can be benign or

ncludes complete and incomplete molar pregnancies (see

**D:** Molar pregnancy may progress to malignant GTD,

**es:** 10–15%.
**noma:** 2–5%.
f malignant GTD include pulmonary or CNS metastases
c pulmonary emboli.

rst-trimester **uterine bleeding,** hyperemesis gravidarum,
ampsia at < 24 weeks, and uterine size greater than dates.

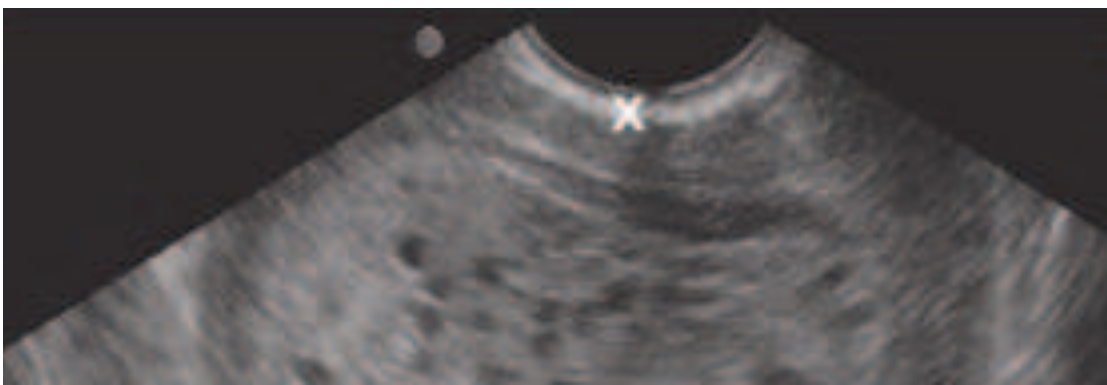
- CXR may show lung metastases.
- D&C reveals **"cluster-of-grapes"** tissue.

### Treatment

- Evacuate the uterus, and follow with weekly β-**hCG.**
- Treat malignant disease with chemotherapy (methotrexate).
- Treat residual uterine disease with hysterectomy.
- Chemotherapy and irradiation are highly effective for

## MULTIPLE GESTATIONS

- Affect 3% of all live births.
- Since 1980, the incidence of monozygotic (identical) births steady, whereas the incidence of dizygotic (fraternal) births has ↑.
- **Hx/PE:** Characterized by rapid uterine growth, excessive gain, and palpation of three or more large fetal parts on
- **Dx:** Ultrasonography; β-hCG, human placental lactogen elevated for GA.
- **Tx:**
  - Multifetal reduction and selective fetal termination higher-order multiple pregnancies.
  - Antepartum fetal surveillance for IUGR.
  - Management by a high-risk specialist is recommended

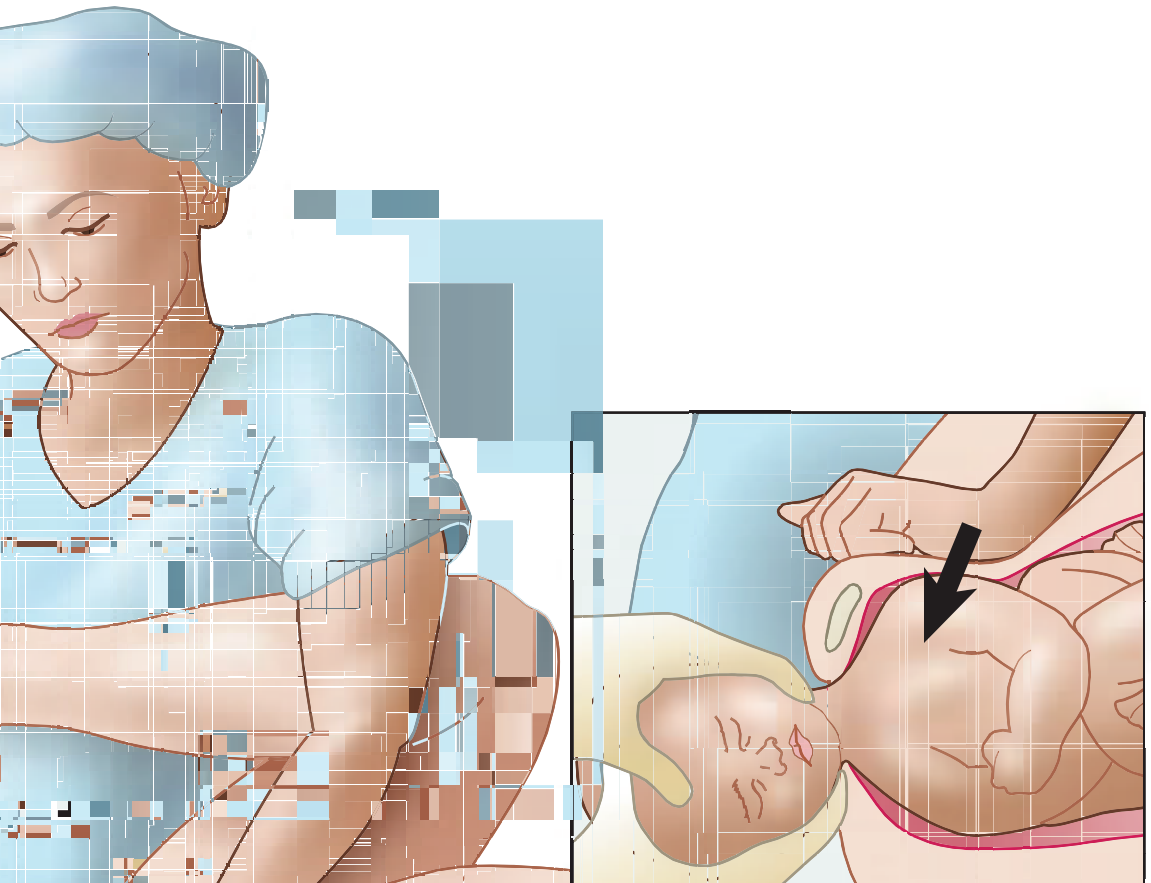


all deliveries in the United States. Risk factors include history of a macrosomic infant, and a history of prior

onged second stage of labor, recoil of the perineum ("tur-
f spontaneous restitution.

cia, be the mother's **HELPER:**

cRoberts maneuver; see Figure 2.11-11).
ubic).



of progressive cervical change.

- **Prolonged second-stage arrest:** Arrest of fetal descent for definitions based on parity and anesthesia.

### Treatment

See Table 2.11-16.

### Complications

- Chorioamnionitis leads to fetal infection, pneumonia,
- Permanent injury occurs in 10%.
- The risk of postpartum hemorrhage is 11%; that of f tion is 3.8%.

## RUPTURE OF MEMBRANES

Distinguished as follows:

- **Spontaneous ROM:** Occurs after or at the onset of lab
- **Premature ROM:** Occurs > 1 hour before onset of lab tated by vaginal or cervical infections, abnormal mem cervical incompetence.

**TABLE 2.11-16. Failure to Progress**

| Stage | definition | treatM |
|---|---|---|
| | **First Stage: Failure to have progressive cervical c** | |
| Latent | ■ **Prima:** > 20 hours<br>■ **Multi:** > 14 hours | Therapeutic rest via parent oxytocin; amniotomy; cerv |
| Active | ■ **Prima:** > 2 hours | Amniotomy; oxytocin; C-se |

**um exam** reveals pooling of amniotic fluid in the vaginal

**r test** is ⊕ (paper turns blue, indicating alkaline pH of

ferning pattern is seen under a microscope after amniotic
lass slide).
to assess amniotic fluid volume.
**s uncertain,** ultrasonography-guided transabdominal instil-
armine dye can be used to check for leakage (unequivocal

ion risk; do not perform digital vaginal exams on women
bor or for whom labor is not planned immediately.
t tracing, maternal temperature, WBC count, and uterine
idence of chorioamnionitis.

To minim
not perf
women

and fetal lung maturity.
check GBS status and fetal presentation; then labor may
r the patient can be observed for 6 hours.
**ks GA:** Labor induction may be considered.
**GA:** Expectant management with bed rest and pelvic rest.
prevent infection and to prolong the latency period in the
ion.
**osteroids:**
thasone or dexamethasone × 48 hours.
al lung maturity in the absence of intra-amniotic infection
eeks GA.
tion or fetal distress develop, give antibiotics (ampicillin
and induce labor.

delivery, chorioamnionitis, placental abruption, cord pro-

fetal testing, or placental abruption.
- **Sterile speculum exam** to rule out PROM.
- **Ultrasonography** to rule out fetal or uterine anomal assess fetal presentation and amniotic fluid volume.
- Obtain cultures for chlamydia, gonorrhea, and GBS urine culture.

### Treatment

- Hydration and bed rest.
- **Tocolytic therapy** (β-mimetics, $MgSO_4$, CCBs, prost unless contraindicated.
- **Steroids** to accelerate fetal lung maturation.
- **Penicillin or ampicillin for GBS prophylaxis** if preter

### Complications

RDS, intraventricular hemorrhage, **PDA,** necrotizing en thy of prematurity, bronchopulmonary dysplasia, death.

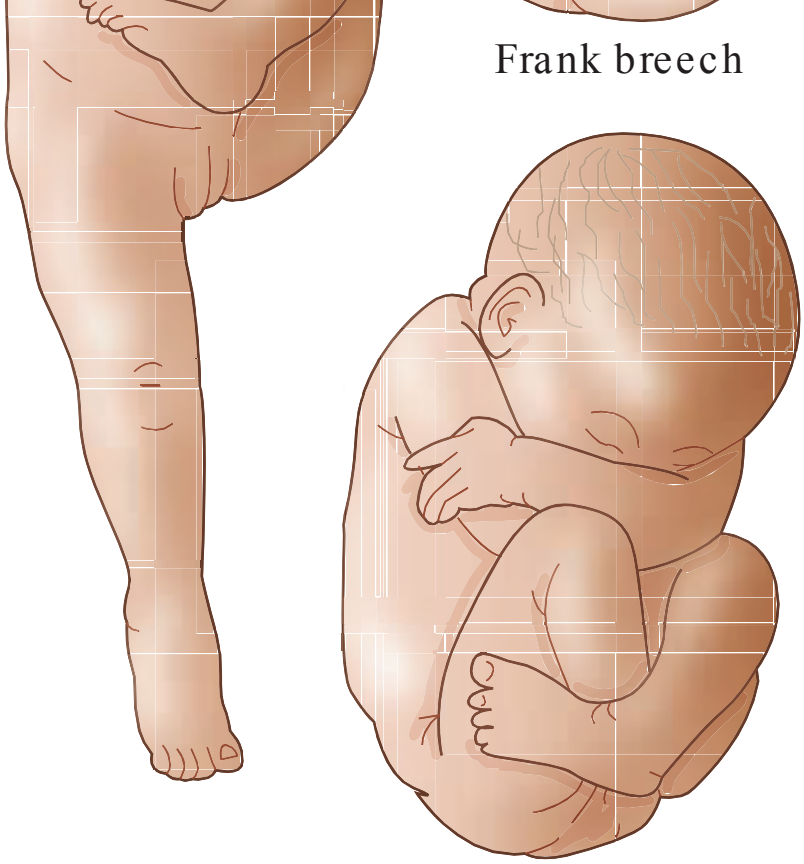
## FETAL MALPRESENTATION

Any presentation other than vertex (ie, head closest t to chest, occiput anterior). Risk factors include **prema** delivery, uterine anomalies, poly- or oligohydramnios, PPROM, hydrocephalus, anencephaly, and placenta previ

### History/Pe

**Breech presentations** are the most common form and i of the fetal lower extremities or buttocks into the materna 2.11-12). Subtypes include the following:

- **Frank breech (50–75%):** The thighs are flexed and the
- **Footling breech (20%):** One or both legs are extended




**Types of breech presentations.** (Reproduced with permission from DeCherney AH. Diagnosis & Treatment, 8th ed. Stamford, CT: Appleton & Lange; 1994.)

## CESAREAN SECTION

for indications. For both elective and indicated cesarean rate should be used in the mother to ↓ gastric acidity and on syndrome.

on of the vaginal opening into the perineum.
(midline) or **mediolateral.**

**dications for Cesarean Section**

- A loss of > **500 mL of blood for vaginal delivery** **C-section.**
- May occur before, during, or after delivery of the place
- Table 2.11-18 summarizes common causes.
- Complications include the following:
  - Acute blood loss (potentially fatal).
  - Anemia due to chronic blood loss (predisposes to pu
  - Sheehan syndrome.
- Severe postpartum hemorrhage may be controlled embolization.

**auses of Postpartum Hemorrhage**

| erine atony | genital tract trauma | retained |
|---|---|---|
| stention (multiple<br>acrosomia,<br>nios).<br>ometrium (rapid<br>l labor, oxytocin<br><br>on.<br>erfering with<br>(anesthesia, myomas, | Precipitous labor.<br>Operative vaginal delivery (forceps, vacuum extraction).<br>Large infant.<br>Inadequate episiotomy repair. | Placenta accreta<br>Placenta previa.<br>Uterine leiomyo<br>Preterm delivery<br>Previous C-secti |
| soft, enlarged, "boggy"<br><br>mon cause of | Manual and visual inspection of the lower genital tract for any laceration > 2 cm long. | Manual and visu<br>placenta and<br>missing cotyle |

**Septic pelvic thrombophlebitis.**
on leads to infection of the vein wall and intimal damage, rn to thrombogenesis. The clot is then invaded by micro-

follows, with liquefaction, **fragmentation, and, finally, ization.**
abdominal and back pain and a "picket-fence" fever c" fevers) with wide swings from normal to as high as F).
h blood cultures and CT looking for a pelvic abscess.
oad-spectrum antibiotics and **anticoagulation** with hepa-ys.

## ME (POSTPARTUM PITUITARY NECROSIS)

ia and necrosis that lead to anterior pituitary insufficiency stetric hemorrhage and shock.

of anterior pituitary insufficiency in adult females.
**mmon presenting syndrome is failure to lactate (due to evels).**
oms include weakness, lethargy, cold insensitivity, genital menstrual disorders.
hormonal testing and MRI of the pituitary and hypothala-umor or other pathology.
t of all deficient hormones. Some patients may recover onadotropin function after cortisol replacement alone.

## REASTFEEDING

cy, ↑ estrogen and progesterone result in breast hypertro-on of the action of prolactin on the breast.

The 7 W
days po

**W**omb (e
**W**ind (at
**W**ater (U
**W**alk (DV
**W**ound (
**W**eaning
masti
**W**onder

Cellulitis of the periglandular tissue caused by nipple trau
ing coupled with the introduction of bacteria, usually Stap
from the infant's pharynx into the nipple ducts.

### History/Pe

- Symptoms often begin 2–4 weeks postpartum.
- Symptoms are **usually unilateral** and include the follo
  - Breast tenderness.
  - A palpable mass.
  - Erythema, edema, warmth, and possible purulent n
- Significant fever, chills, and malaise can also be seen.

### Diagnosis

- Differentiate from simple breast swelling.
- Infection is suggested by focal symptoms, an ↑ WBC c

### Treatment

- **Continued breastfeeding** to prevent the accumulatio
  rial (or use of a breast pump in patients who are no lon
- **PO antibiotics** (dicloxacillin, cephalexin, amoxicillin/
  mycin, clindamycin).
- If not clinical improvement within 48–72 hours, evalu
  sonography to assess for abscess. If present, treat with
  age.

Female Development 332
e 332
333
334
Menstrual Cycle 334
Puberty 334
338
340
340
ING 341
ology 343
PERPLASIA 343
d Ro ME 345
346
s 347
RTHo LIN d Uc T 347
348
349
BEASE 349
350
Gynecologic Neoplasms
Uterine Leio myo ma (Fibro id s)
End o metrial c anc er
c ervic al c anc er
Vulvar c anc er
Vaginal c anc er
o varian c anc er
Pelvic Organ Prolapse
Urinary Incontinence
Pediatric Gynecology
Ped iatric Vaginal d isc harge
Prec o c io us Puberty
Benign Breast Disorders
No nPro liferative Breast Lesio ns
Pro liferative Breast Lesio ns Witho u
Fibro Ad eno ma
Phyllo d es Tumo r
Atypic al Hyperplasia
Breast Cancer
Sexual Assault

# Normal Menstrual Cycle

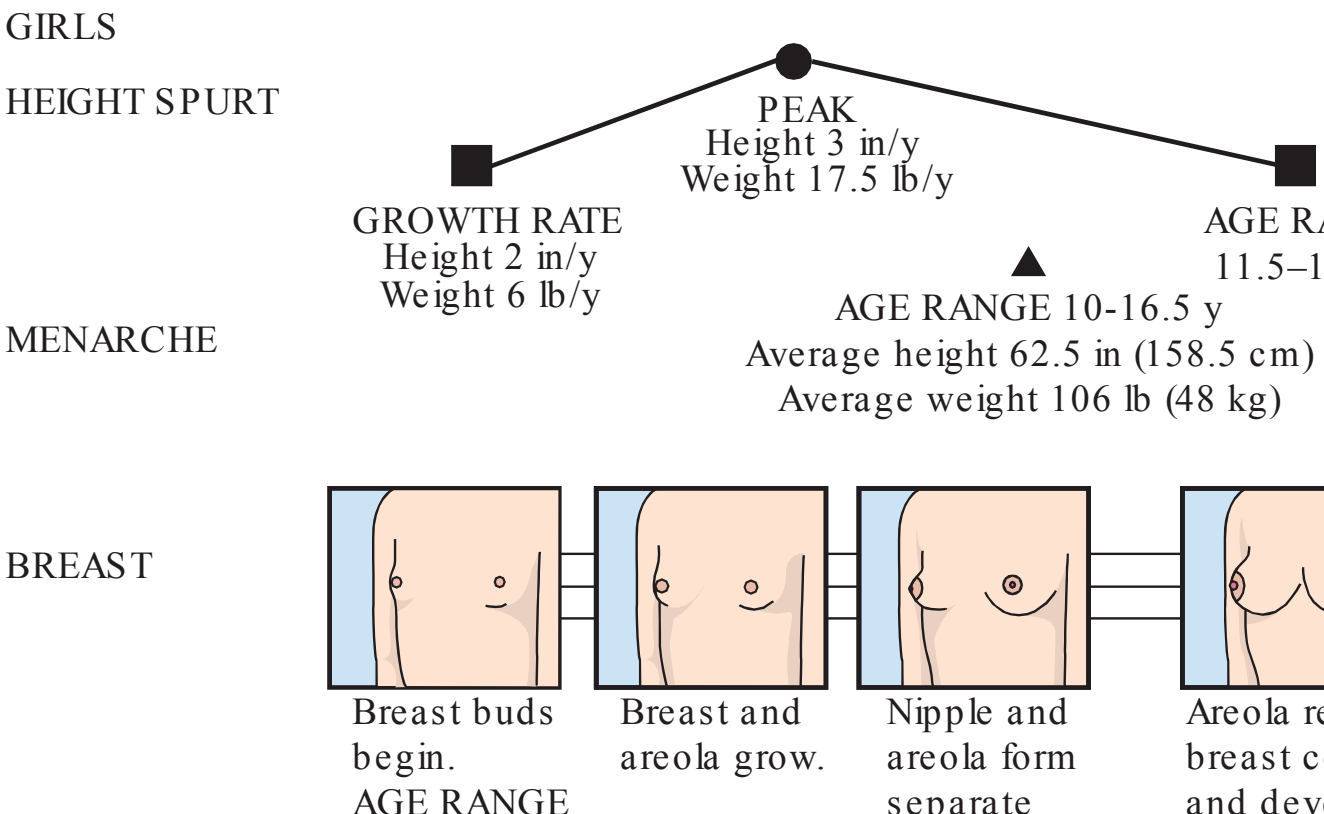
The progression of a normal menstrual cycle is as follo
2.12-2):

**Menstruation/follicular phase (days 1–13):**

- Starts with menstruation and ends at LH surge/ovulatio
- May vary, but typically lasts ~ 13 days.
- ↑ frequency of GnRH pulse → ↑ FSH → growth of fo
  production.
- Results in the development of straight glands and th
  uterine lining (proliferative phase).
- By late follicular phase: Dominant follicle is selected a
  uterine endometrium thickens, and cervical mucus be

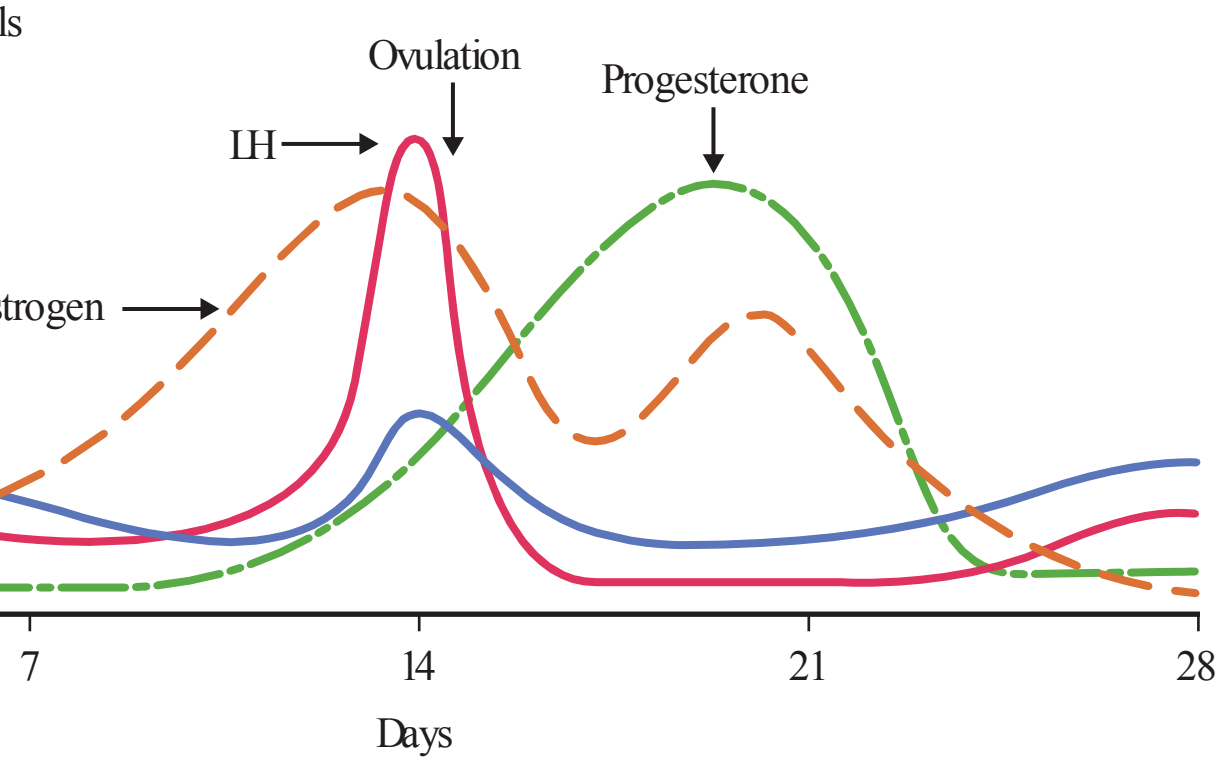
**Ovulation (day 14):**

- Estradiol reaches a peak → positive feedback to the pi
  surge (smaller FSH rise) → rupture of the ovarian foll
  mature ovum → travels to oviduct/uterus.
- Ruptured follicular cells differentiate into the corpus lu



roliferative phase
(follicular)

Secretory phase
(luteal)




**rmal menstrual cycle.** (Adapted with permission from USMLE-Rx.com as published in Step 1 2015. New York, NY: McGraw-Hill; 2015.)

**15–28):**

me (10–14 days) that the corpus luteum can survive without hCG stimulation.

strogen to progesterone predominance: Corpus luteum terone and some estradiol, allowing the endometrial lining thick and tortuous endometrial glands with thick secretions .

of fertilization and implantation, ↓ LH → ↓ progesterone the corpus luteum → results in sloughing of the endome-

and progesterone there is no longer negative feedback to n increases and restarts menstruation/follicular phase.

- May ↑ cardiovascular morbidity and mortality.
- **Contraindications:** Vaginal bleeding, breast can
  pected), untreated endometrial cancer, a history of
  chronic liver disease, and hypertriglyceridemia.

- **Non-HRT:** SSRI/SNRIs, clonidine, and/or gabapentin
  of hot flashes.
- **Topical estrogen preparation:** For **vaginal atrophy.** I
  same contraindications as systemic HRT.
- **Supplements ± bisphosphonates:** For **osteoporosis.** D
  measure bone mineral density (BMD). Supplemental
  daily calcium/vitamin D and weight-bearing exercise.

## Contraception

Eighty-five percent of sexually active women with no
become pregnant within 1 year.

- Table 2.12-1 describes the effectiveness of contracep
  with their relative advantages and disadvantages.
- See Table 2.12-2 for contraindications to common me
  tion.
- Emergency contraception (EC) methods prevent pre
  tected sex or contraceptive failure. Table 2.12-3 de
  methods of EC.

## Abnormalities of the Menstrual Cycle

### 1° AMENORRHEA/DELAYED PUBERTY

The absence of menses by age 16 with 2° sexual develop

| | | | |
|---|---|---|---|
| | Foreign body results in inflamm tion; progesterone leads to cervical mucus thickening and endometrial decidualization. | Ef ective for up to 5 years.<br>Immediate fertility once removed.<br>Safe with breastfeeding.<br>Lighter periods; less cramping. | Sp<br>Ris<br>In r |
| | Foreign body results in inflamm tion; copper has a spermicidal ef ect.<br>No hormonal exposure. | Ef ective for up to 10 years.<br>Immediate fertility once removed.<br>Safe with breastfeeding. | ↑ c<br>Ris<br>In r |
| | | Permanently ef ective; safe with breastfeeding. | **Tu**<br>p<br>**Va**<br>t<br>s |
| | **Very effective: 90–99%** | | |
| e) | IM injection (progestin). | Lighter or no periods.<br>Each shot works for 3 months.<br>Safe with breastfeeding. | Irre<br>De<br>De<br>c<br>r |
| ") | Combined weekly estrogen and progestin dermal patch. | Periods may be more regular.<br>Weekly administration. | Th<br>in s |
| | Combined low-dose progestin and estrogen vaginal ring. | Can make periods more regular.<br>3 weeks—continuous; 1 week—no ring.<br>Safe to use continuously. | Ma<br>Sp |

| | | |
|---|---|---|
| prevent entry of sperm. | | |
| arrier sheath is inserted into the ina. | Some protection against STDs. | Can be diffic |
| ual intercourse is avoided on s in the menstrual cycle on ch conception is likely (near the e of ovulation). | No side effects. | Requires the<br>No STD/HIV |
| **less effective: 68–74%** | | |
| penis is removed prior to culation. | No side effects. | No STD/HIV<br>Not recomm |
| ubstance that inhibits sperm tility. | May be used as a 2° method. | Not recomm |

hods (eg, patch, ring) may also protect against endometrial and ovarian cancer; howeve duction.

**TABLE 2.12-2. Contraindications to Common Methods of Contra**

| Estrogen-Containing Hormonal Methods[a] | IUDs (Progester |
|---|---|
| Pregnancy | Severe uterine structural |
| History of stroke, HTN, DVT/PE | (bicornuate, septate) |
| Breast cancer | Known or suspected pre |
| Undiagnosed abnormal vaginal bleeding | Active gynecologic infec |
| Estrogen-dependent cancer | Unexplained vaginal/ute |

EC.

| | | |
|---|---|---|
| | Can be used as EC and continued for up to 10 years of contraception. | High initial cost of in<br>Must be inserted by<br>No protection again<br>Must test for pregna |

of unprotected sex.
nprotected sex.

syndrome (isolated gonadotropin deficiency) associated
**mia.**
**ional growth delay.**
**exual characteristics** (estrogen production but other ana-
problems): Etiologies include the following:
**genesis:** Absence of two-thirds of the vagina; uterine
s.
**hymen:** Presents with hematocolpos (blood in the vagina)
scape, along with a bulging hymen.
**ndrogen insensitivity:** Patients present with breast devel-
matization of testosterone to estrogen) but are amenor-
k pubic hair.
**adrenal hyperplasia:** Can present as virilization with
or oligomenorrhea.

The first
amenor

**y test.**
Pubertal development, genital exam, signs of androgen
features of Turner syndrome.
mic abnormalities (imperforate hymen): Physical exam,

## 2° AMENORRHEA

The absence of menses for 6 consecutive months in wom
menarche. Etiology includes:

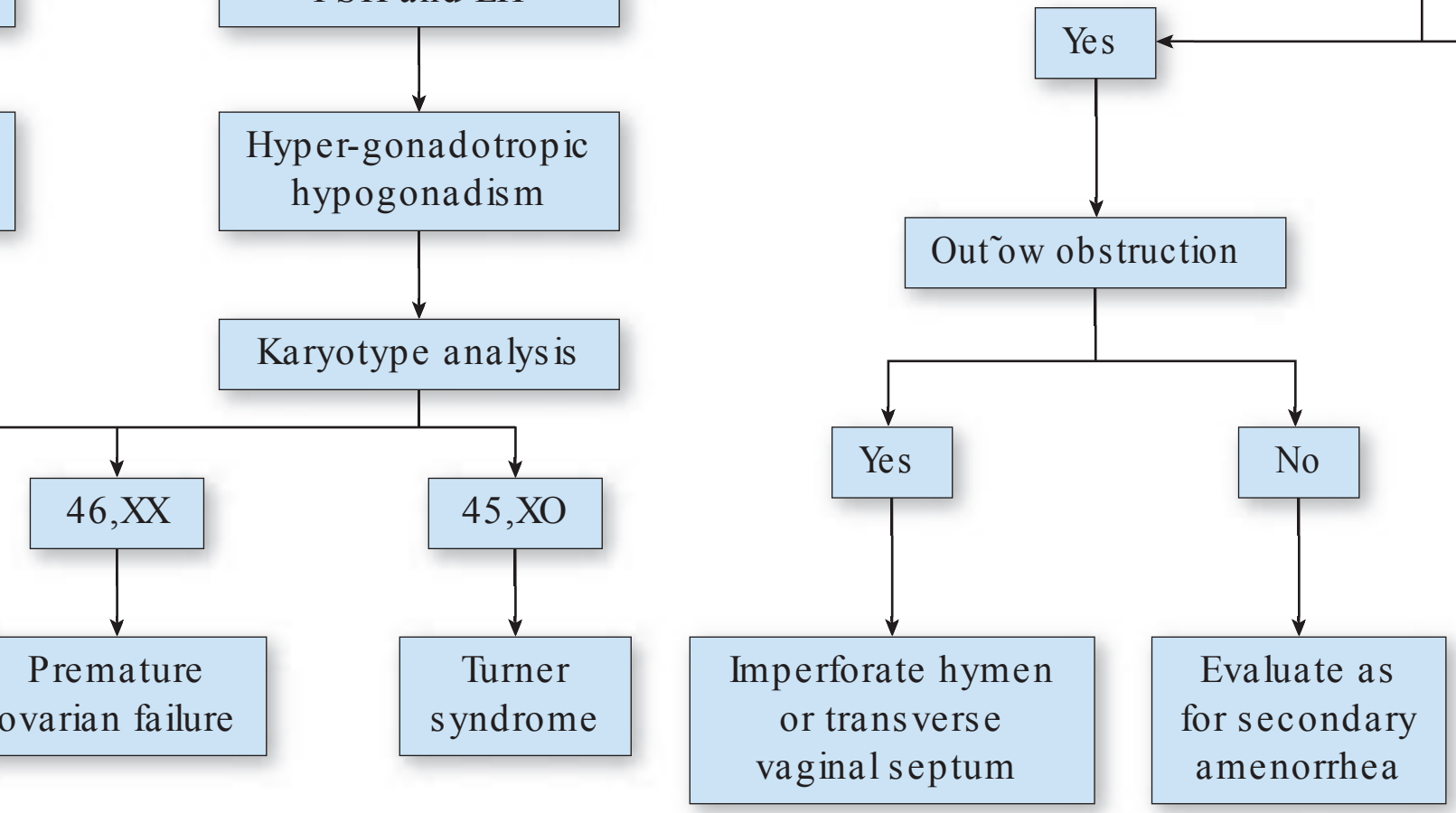
- **Pregnancy.**
- **Ovary:** PCOS, premature ovarian failure.
- **Hypothalamus:** Neoplasm, functional hypothalamic
  tion, exercise, and stress), systemic illness (diabetes me
  celiac disease).
- **Pituitary:** Adenoma (eg, prolactin secreting), sellar m
  drome.
- **Thyroid:** Hypothyroidism.
- **Uterine:** Asherman syndrome, cervical stenosis.

### Diagnosis

- History and physical exam.
- Get a pregnancy test.
- If ⊖, measure TSH and prolactin.
- ↑ **TSH:** Indicates hypothyroidism.
- ↑ **prolactin** (inhibits the release of GnRH and thus L
  to a pituitary pathology. Order an MRI of the pituitary
  tin-secreting pituitary adenoma.

**TABLE 2.12-4. Etiologies of 1° Amenorrhea**

| | GnRH | LH/FSH | Estrogen/Progesterone | |
|---|---|---|---|---|
| Constitutional growth delay | ↓ | ↓ | ↓ (prepuberty levels) | Pu |



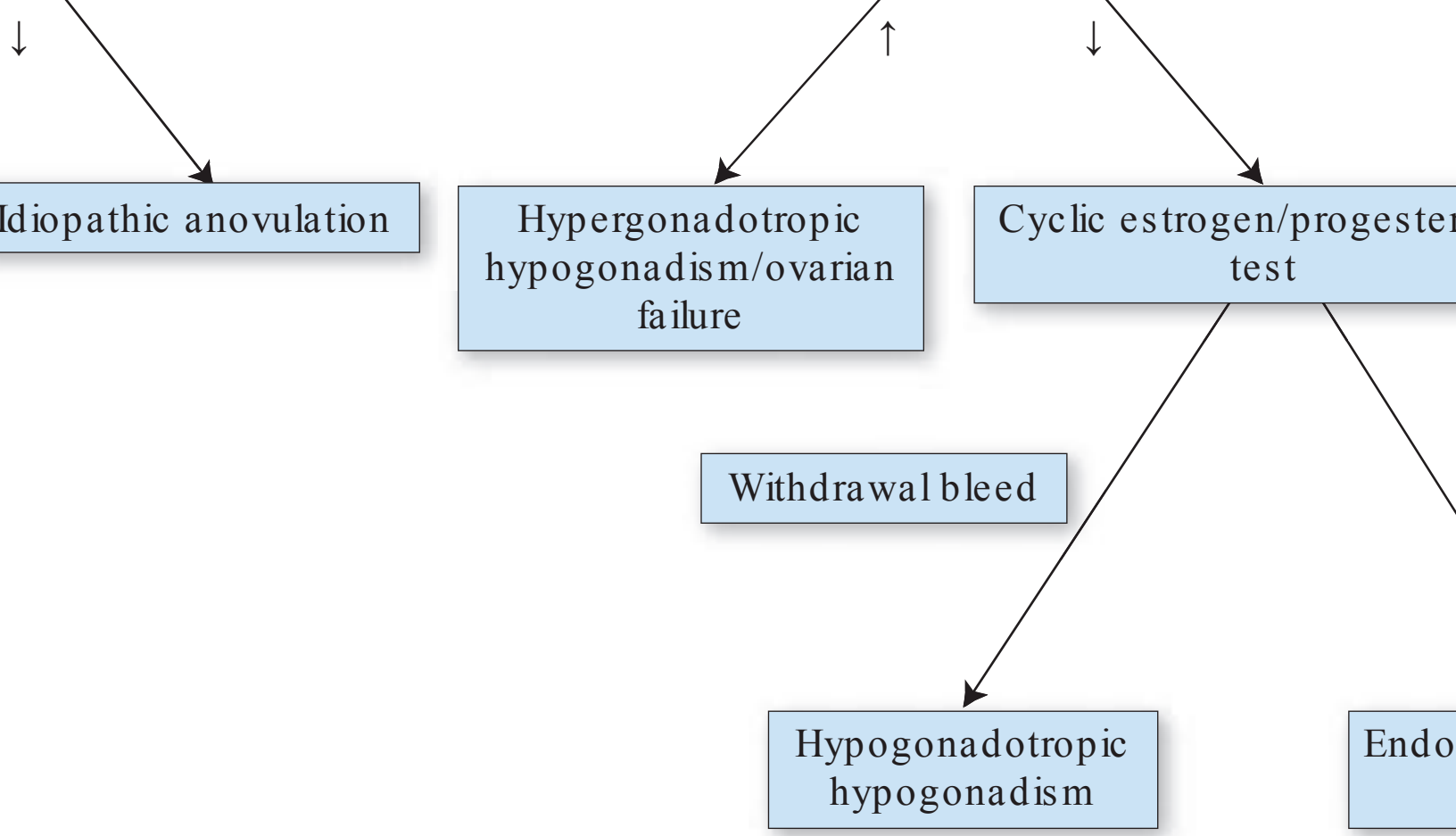

**orkup of 1° amenorrhea.** (Modified with permission from DeCherney AH et al. Current Diagnosis &Treat .)

**tin challenge** (10 days of progestin).
4 for an algorithm of the diagnostic workup.
**allenge (withdrawal bleed):** Indicates anovulation that is noncyclic gonadotropin secretion, pointing to PCOS or lation.
**llenge (no bleed):** Indicates uterine abnormality or estro-

**lycemia (polydipsia, polyuria) or hypotension:** Conduct nt dexamethasone suppression test to distinguish CAH deficiency), Cushing syndrome, and Addison disease.
**zation is present:** Measure testosterone, DHEAS, and sterone.
COS, CAH, or Cushing syndrome.
**ere pattern:** Look for an ovarian or adrenal tumor.

Q

A 56-y
compl




**2° amenorrhea.**

## 1° DYSMENORRHEA

Menstrual pain associated with ovulatory cycles in the ab findings. Caused by uterine vasoconstriction, anoxia, an tions mediated by an excess of prostaglandin ($PGF_{2\alpha}$).

### History/Pe

- Presents with low, midline, spasmodic pelvic pain that back or inner thighs.
- Cramps occur in the first 1–3 days of menstruation an with nausea, diarrhea, headache, and flushing.
- There are **no pathologic findings on pelvic exam.**

### Diagnosis

ferential to rule out infection or neoplasm.
t UTI.
chlamydial swabs to rule out STDs/PID.
athology causing pain (see Table 2.12-5).

y specific.

## NE BLEEDING

al bleeding ranges from 2 to 7 days. Abnormal uterine
o alterations in quantity, duration, or frequency. Classified
M-COEIN.

**ometriosis vs Adenomyosis**

| endo Met rio SiS | a de |
|---|---|
| ctional endometrial glands and stroma **outside** the rus. | Endometrial tissue **in** the m |
| **lic** pelvic and/or rectal pain and dyspareunia. Uterus enlarged but **painful nodules, restricted range of tion.** | Classic triad of pain, **menor** **boggy, symmetric** uterus. |
| uires direct visualization by **laparoscopy** or laparotomy.<br>ssic lesions have a blue-black ("raspberry") or dark rown ("powder-burned") appearance.<br>ovaries may have endometriomas ("chocolate cysts"). | **MRI** can aid in diagnosis bu<br>Ultrasonography is useful b<br>between leiomyoma and<br>Ultimately a pathologic diag |
| **rmacologic:** Inhibit ovulation. Combination OCPs (first | **Pharmacologic:** Largely sy |

- **Menorrhagia:** ↑ amount of flow (> 80 mL of blood lo
  longed bleeding (flow lasting > 8 days); may lead to an
- **Metrorrhagia:** Bleeding between periods.
- **Menometrorrhagia:** Excessive and irregular bleeding.
- On pelvic examination, look for an enlarged uterus,
  polyps to assess for myomas, pregnancy, or cervical can

### Diagnosis

- **β-hCG:** To rule out pregnancy.
- **CBC:** To evaluate for anemia.
- **Pap smear:** To rule out cervical cancer.
- **Gonorrhea/chlamydia probe:** To rule out cervical bl
  tis.
- **TFTs and prolactin**: To rule out hyper-/hypothyroidis
  tinemia.
- **Platelet count, PT/PTT:** To rule out von Willebran
  XI deficiency, primarily in adolescent patients.
- **Ultrasonography:** To look for uterine masses, poly
  thickness of the endometrium.
- Endometrial biopsy should be performed:
  - If the endometrium is ≥ 4 mm in a **postmenopausa**
  - If the patient is > 35 years of age with risk fact
    hyperplasia (eg, obesity, diabetes).

### Treatment

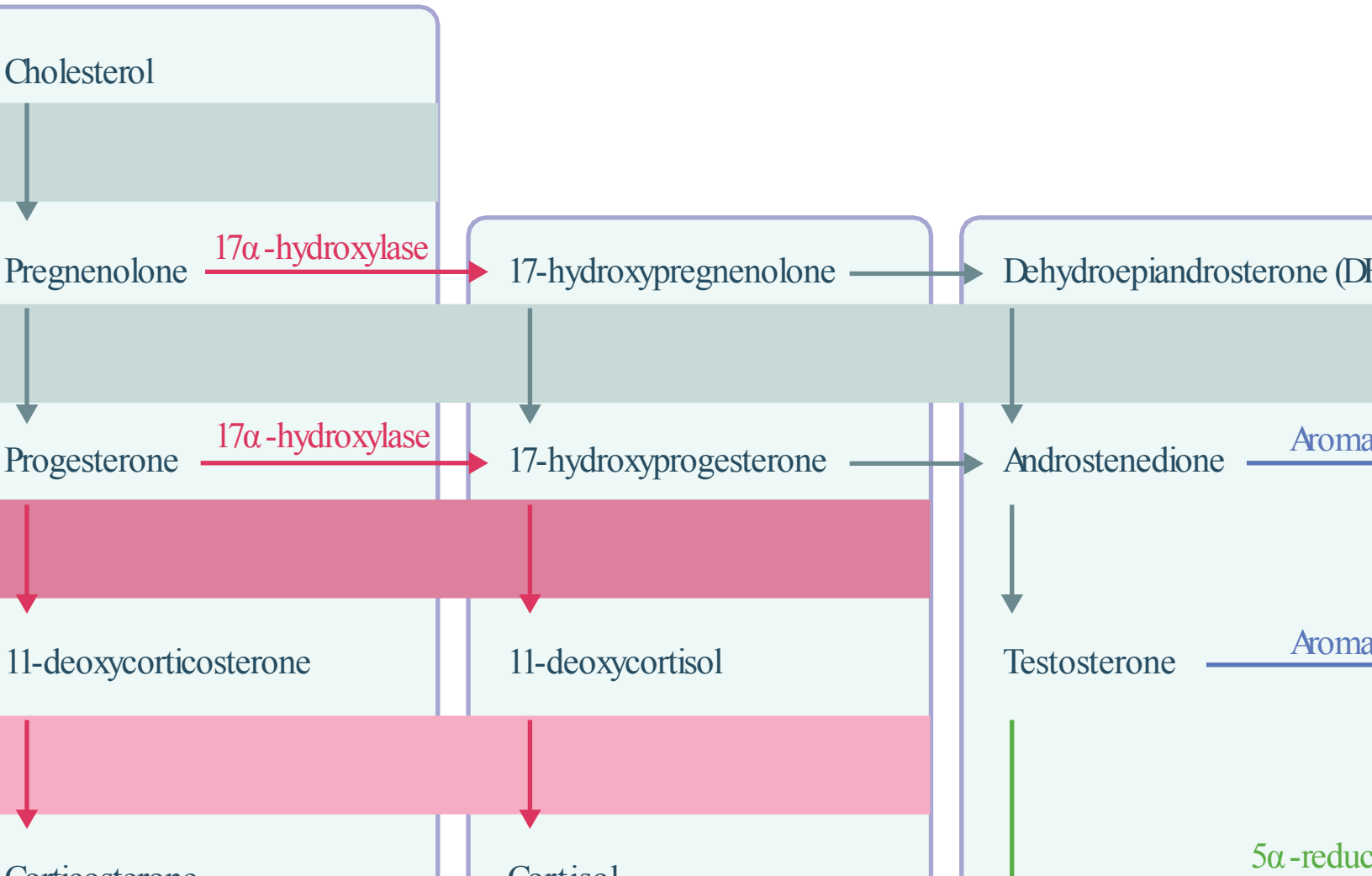
- **Acute heavy bleeding:**
  - High-dose estrogen IV stabilizes the endometrial
    stops bleeding within 1 hour. Transition to combi
    tive or add progestin when bleeding stabilized.
  - If estrogen is contraindicated, can give high dos
    alone.
  - If bleeding is not controlled within 12–24 hours, a
    cated.

# ctive Endocrinology

## NAL HYPERPLASIA

ast one enzyme required for the biochemical synthesis of sterol (see Figure 2.12-5 and Table 2.12-6). Includes the

**deficiency:** Accounts for ~ 90% of CAH cases; "classic" vere and presents as a newborn infant girl with ambigu- d adrenal insufficiency (with or without life-threatening Nonclassic" is a late-onset form that presents with andro-



| | | | | | | |
|---|---|---|---|---|---|---|
| | ↓ | ↑ | ↓ | ↓ | ↑ renin activity<br>↑ 17-hydroxy-progesterone | Mo<br>Pre<br><br>XX |
| erone<br>oxycorti-<br>rone (results<br>P) | ↓ | ↑ | ↑ | ↓ | ↓ renin activity | XX |

eficiencies a e characterized by an enlargement of both adrenal glands due to ↑ ACTH s

n Le T et al. First Aid for the USMLE Step 1 2015. New York, NY: McGraw-Hill; 2015.

gen excess or could be asymptomatic. Cannot conver terone to 11-deoxycortisol → ↓ cortisol synthesis → ↑ → ↑ ACTH and androgens.

- **11β-hydroxylase deficiency:** Second most common hyperplasia. Cannot convert 11-deoxycortisol to cortis costerone to corticosterone, also leading to ↑ ACTH an

n.

nd

ze; loss

### History/Pe

Androgen excess: Genital ambiguity, premature pubarch larity, infertility, hirsutism, acne, and, rarely, a palpable pe

### Diagnosis

- Physical exam.
- **21-hydroxylase deficiency: ↑ 17-OH progesterone**

nce of 6–10% among US women of reproductive age and
cause of infertility in women. Syndrome of excess testos-
strogen. Diagnosis requires fulfillment of two of the fol-
dam criteria):

s (via ultrasonography).
ovulation.
biochemical evidence of hyperandrogenism.

ion: Obesity (BMI > 30 kg/m$^2$), menstrual cycle distur-
acne, androgenic alopecia, and hirsutism from hyperan-

are also at ↑ risk for the following:

s nigricans.
ome—insulin resistance, atherogenic dyslipidemia, and

**ting of hyperandrogenemia:** ↑ testosterone.
erone more sensitive than total testosterone (total can be
to low sex hormone-binding globulin.
**auses of hyperandrogenism:**
ule out adrenal tumor.
und to rule out androgen secreting ovarian tumor.
sterone to rule out nonclassical CAH.
eening in the setting of clinical signs of Cushing syn-
oon facies, buffalo hump, abdominal striae) or acromeg-
d size).
**tabolic abnormalities:**
l glucose tolerance test.
and lipoprotein levels (total cholesterol, HDL, LDL, tri-

The mos
PCOS is
**H**yper**A**
and **Aca**



**FIGURE**
**with pro**
duced with
Current Dia
10th ed. Ne

A 28-y
for a w
that ap
mense

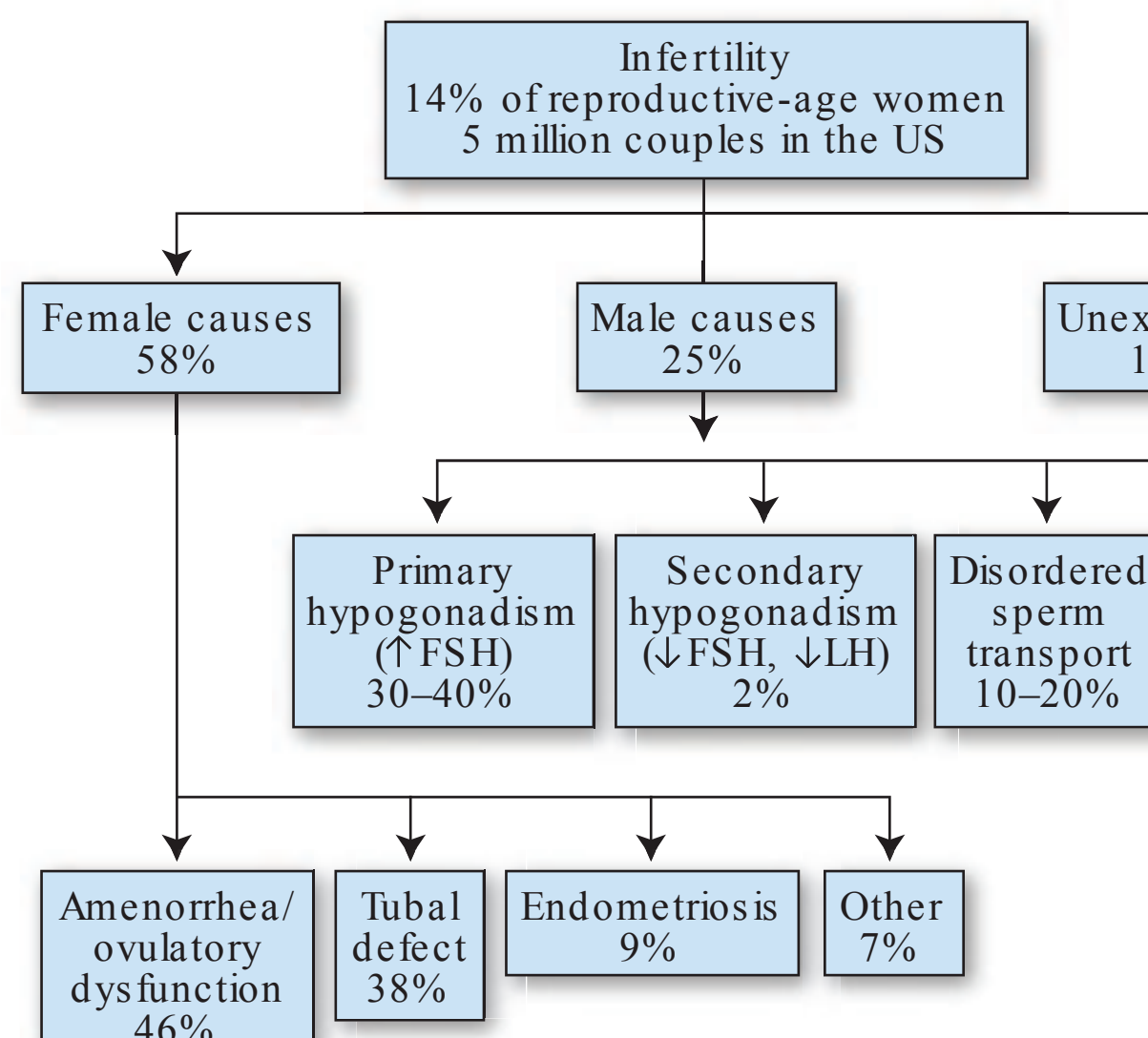
controlling medication (eg, statins).

### Complications

↑ risk of early-onset DM2; ↑ risk of miscarriages; ↑ long
and endometrial cancer due to unopposed estrogen secret

## INFERTILITY

- The inability to conceive after 12 months of normal, r
  sexual activity.
- 1° infertility is characterized by no prior pregnancies;
  in the setting of at least one prior pregnancy. Etiologi
  ure 2.12-7 and Table 2.12-7.



| | | |
|---|---|---|
| Age (incidence ↑ with age).<br>Symptoms of hyper-/ hypothyroidism.<br>Galactorrhea.<br>Menstrual cycle abnormalities. | Menstrual history.<br>Basal body temperature.<br>Ovulation predictor.<br>Midluteal progesterone.<br>Early follicular FSH ± estradiol level (ovarian reserve).<br>TSH, prolactin, androgens.<br>Ovarian sonography (antral follicle count).<br>Endometrial biopsy (luteal-phase defect). | Treat<br>(eg<br>Indu<br>**clo**<br>pu<br>IUI.<br>IVF. |
| History of PID, appendicitis, endometriosis, pelvic adhesions, tubal surgery. | Hysterosalpingogram.<br>Endometrial biopsy. | Lapa<br>en<br>IVF. |
| Cryotherapy, conization, or DES exposure in utero. | Pap smear.<br>Physical examination.<br>Antisperm antibodies. | IUI w<br>IVF. |
| Polyps.<br>Fibroids.<br>Congenital anomalies. | Ultrasound.<br>Sonohysterogram.<br>Hysterosalpingogram. | Surg |

# ogic Infections

## OF BARTHOLIN DUCT

burning, irritation, and abnormal discharge. The most c
bacterial vaginosis, vulvovaginal candidiasis, and tricho
2.12-8).

**ginitis**

| **cterial Vaginosis** | **Trichomonas** | |
|---|---|---|
| st common). | 5–50%. | 15–30%. |
| tion: shift in vaginal flo a<br>s, ↓ lactobacilli). | Protozoal flagell tes **(an STD).** | Usually Candida |
| nultiple sexual partners,<br>l partner, frequent | Unprotected sex with multiple partners. | **DM, antibiotic**<br>**corticosteroid**<br>↑ frequency of<br>fitting clothin |
| harge. | ↑ discharge, odor, pruritus, dysuria. | Pruritus, dysuri |
| rritation. | "Strawberry petechiae" in the upper vagina/cervix. | Erythematous,<br>vagina. |
| us, **grayish-white, fis y**/ | Profuse, malodorous, **yellow-green, frothy.** | Thick, white, cu<br>odor. |
| (epithelial cells coated<br>; see Figure 2.12-8); "No<br>nell fish in the agina | **Motile trichomonads** (flagell ted organisms that are slightly larger than WBCs). See Figure 2.12-8. | — |
| st (fis y smell) | — | **Pseudohyphae** |

B

**uses of vaginitis. (A) Candidal vaginitis.** Candida albicans organisms are evident o
he granular epithelial cells ("clue cells") and indistinct cell margins. **(C)** Trichomon
"strawberry cervix." (Part A reproduced with permission from Wolff K et al. Fitzpatrick's Color Atlas & Synop
B reproduced with permission from USMLE-Rx.com. Part C reproduced courtesy of the US Department of Hea

hange in discharge, malodor, pruritus, irritation, burning,
unia, and dysuria.
ns are as follows:
**rogen surge:** Clear, elastic, mucoid secretions.
**e/pregnancy:** Thick and white secretions; adhere to the

ugh examination of the vulva, vaginal walls, and cervix.
y WBCs and no organism on saline smear, suspect Chla-

vaginal pH, amine (whiff) test, wet mount (with saline),
um hydroxide (KOH) microscopy.
arge, numerous leukocytes on wet prep, cervical friability,
ns of PID, order DNA tests or cultures for Neisseria gonor-
ydia trachomatis to rule out cervicitis.
logy specific (see Table 2.12-8).

**f the uterine cervix.** Etiologies are as follows:
most common): Chlamydia, gonococcus, Trichomonas,
ydia trachomatis is more common than gonococcus in
t cervicitis.

Criteria
bacteria
required
- Abn
- Vagi
- ⊕ a
- Clue
epit

Genital
cause si
Haemop
painful u

one of the following:
- Uterine tenderness.
- Adnexal tenderness.
- Cervical motion tenderness.

- First, order a β-hCG to rule out pregnancy.
- A WBC count > 10,000 cells/μL has **poor positive a tive value** for PID.
- Ultrasonography may show thickening or dilation of fluid in the cul-de-sac, a multicystic ovary, or tubo-ovar

### Treatment

- Antibiotic treatment should not be delayed while awa All sexual partners should be examined and treated app
- **Outpatient regimens:**
  - **Regimen A:** Ceftriaxone IM × one dose **or** cefoxi **plus** doxycycline × 14 days ± metronidazole × 14 da
  - **Regimen B:** Ofloxacin **or** levofloxacin × 14 days ± days. Only in special cases given ↑ quinolone-resist
- **Inpatient antibiotic regimens:**
  - Cefoxitin or cefotetan plus doxycycline × 14 days.
  - Clindamycin plus gentamicin × 14 days.
- **Surgery:**
  - Drainage of a tubo-ovarian/pelvic abscess is appropr sists after antibiotic treatment; the abscess is > 4–6 the cul-de-sac in the midline and drainable through
  - If the patient's condition deteriorates, perform exp or laparotomy.
  - Surgery may range from TAH/BSO with lysis of cases to conservative surgery for women who desir ity.

### Complications

- Repeated episodes of infection, chronic pelvic pain

**ormed toxin** and not invasive properties of the organism.

**ion,** examination for foreign objects in vaginal canal, zed infection found.
**tics:** Clindamycin + vancomycin.
nsitive S aureus isolated in wound: Clindamycin + oxacil-

sistant S aureus isolated: Clindamycin + vancomycin OR

te associated with TSS is 3–6%.
include cardiac arrhythmias, cardiomyopathy, respiratory RDS, and coagulopathy due to DIC.

## ogic Neoplasms

s include uterine, endometrial, ovarian, cervical, and vul-ian cancer carries the highest mortality.

### MA (FIBROIDS)

**benign** neoplasm of the female genital tract. They pre-nd, firm, and often multiple tumors composed of smooth tive tissue.

mone sensitive, size will ↑ in pregnancy and ↓ after meno-

ormation to leiomyosarcoma is rare (0.1–0.5%).
e common in black (50%) than white (25%) women.

Uterine
cause in
words o
irregular

- **If asymptomatic:** Expectant management with annua CBCs as needed.
- **Pharmacologic:**
  - Combined hormonal contraception.
  - Medroxyprogesterone acetate or danazol to slow or
  - GnRH analogues (leuprolide or nafarelin) to ↓ t suppress further growth, and ↓ surrounding vascula to surgery.
  - NSAIDs for pain.
- **Surgery:**
  - **Women of childbearing years:** Abdominal or hys tomy.
  - **Women who have completed childbearing:** Total nal or vaginal hysterectomy.
  - **Uterine artery embolization** (~ 25% will need fu ment).
  - Emergent surgery may be required if torsion myoma.

### Complications

Infertility may be due to a myoma that distorts the uterin role similar to that of an IUD.

## ENDOMETRIAL CANCER

Type I endometrioid adenocarcinomas derive from **at hyperplasia** and are the most common female reprodu United States. Type II cancers derive from serous or clea Table 2.12-9). Although type II cancers tend to be more a and management are similar for both types.

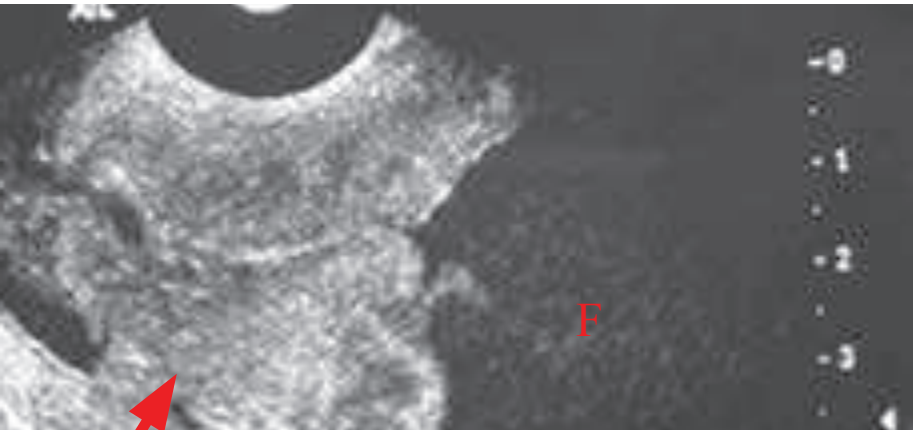
### History/Re

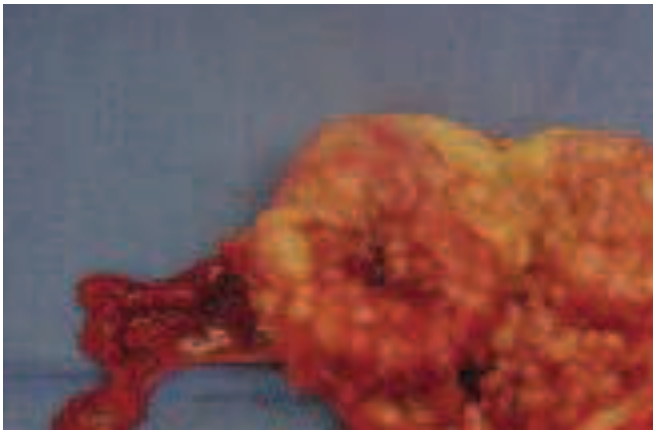
| | | |
|---|---|---|
| | Hyperplasia and atypical hyperplasia. | None. |
| s | 55 years. | 67 years. |
| | Favorable. | Poor. |

stins for women of childbearing age.
iation for postmenopausal women.
adjuvant chemotherapy for advanced-stage cancer.

s proximal to the external os, is non-visible, and is com-
cells (similar to the lower uterine segment). The ectocer-
omposed of squamous cells (similar to the vagina). The
ar cells to an acidic vaginal pH results in metaplasia to
he normal squamocolumnar junction (transformation
the ectocervix and can be exposed to carcinogens, result-
epithelial neoplasia (CIN), an abnormal proliferation or
asal cell layer.

When to
endome
- Post
spot
- Pren
inte
mer
initi
uno



common signs.
- A bloody or purulent, malodorous, nonpruritic discha invasion.

### Screening

- The American Congress of Obstetricians and Gyn currently recommends that screening for cervical can regardless of onset of sexual activity.
- Screening recommendations for women with previous
  - < 21 years: No screening.
  - 21–29 years: Every 3 years perform Pap smear (cyto
  - 30–65 years: Every 3 years perform Pap smear **or** e Pap smear or every 5 years perform co-testing (Pap
  - 65 years: Stop screening if prior tests were negative.
  - See Table 2.12-10 for classification systems of Pap s

Women with DES exposure (risk for clear cell cancer) a promised status (including HIV positivity) should continu long as they do not have a life-limiting condition.

### Diagnosis

The diagnosis and follow-up of specific subtypes of cer be as follows (these guidelines do not apply to adoles women). Cotesting (HPV test and Pap smear) is routinely years.

1. Women 21–24 years with cervical cytology showing **cells of undetermined significance (ASC-US) or lo intraepithelial lesion (LSIL).**
   - **Repeat cytology at 12 months (preferred):**
     - **If ⊖, ASC-US, or LSIL:** Repeat cytology at 12 then routine screening but if ASC or higher the
     - **If ASC-H, HSIL, atypical glandular cells:** Colp

with cervical cytology showing AGC should have colpos-
docervical sampling.
years of age or those with abnormal bleeding raising con-
metrial neoplasia should have colposcopy with endocervi-
metrial sampling.
**L in 21–24 years:** Colposcopy and then proceed as follows:
**:** Treatment as below.
**, III:** Colposcopy and cytology at 6 month intervals for 2

then routine screening.
**1 year:** Biopsy.
**4 years:** Immediate loop electrosurgical excision (not if
poscopy.

**sease,** treatment based on biopsy results for noninvasive
ase) is as follows:

by ASC-US, LSIL, or HPV ⊕.
y of treatment is **follow-up.**
12 months (or cytology if < 30).
higher or HPV ⊕: Colposcopy.
ests: Resume routine screening.
by ASC-H or HSIL.
tology if < 30) at 12 and 24 months.
**cytology** ⊖**:** Resume routine screening.
**abnormal cytology** other than HSIL: Colposcopy.
gnostic excision procedure (unless 21–24 or pregnant).

ion procedure (unless 21–24 years or pregnant).
**4:**

Prognosis

- The overall 5-year relative survival rate for carcinoma in Caucasian women and 55% in black women.
- Survival rates are inversely proportionate to the stage o
  - **Stage 0:** 99–100%.
  - **Stage IA:** > 95%.
  - **Stage IB–IIA:** 80–90%.
  - **Stage IIB:** 65%.
  - **Stage III:** 40%.
  - **Stage IV:** < 20%.
- Almost two-thirds of patients with untreated carcinoma uremia when ureteral obstruction is bilateral.

## VULVAR CANCER

Risk factors include **HPV (types 16, 18, and 31), lichen s** medical exams, diabetes, obesity, hypertension, cardiovas ing, high-risk sexual behavior, and immunosuppression. V neoplasia (VIN) is precancerous and is more commonly pausal women.

| Stage | 0 | I | II | III |
|---|---|---|---|---|
| Extent of tumor | Carcinoma in-situ | Conÿned to cervix | Disease beyond cervix but not to pelvic wall or lower 1/3 of vagina | Disease to pelvic wall or lower 1/3 of vagina |
| 5-year survival | 100% | 85% | 65% | 35% |
| Stage at presentation | | 47% | 28% | 21% |



nopausal women.

**:** Topical chemotherapy, laser ablation, wide local excilvectomy, and simple vulvectomy.

ctomy and regional lymphadenectomy, or
xcision of the 1° tumor with inguinal lymph node dissecrative radiation, chemotherapy, or both.

of all gynecologic malignancies. Risk factors include , chronic irritation (eg, long-term pessary use or prolapse low socioeconomic status, radiation for cervical cancer, splasia, multiple sexual partners, and DES exposure. Etis:

**women:** Usually squamous cell carcinoma.
**n:** Usually other histologic types (eg, adenocarcinoma, carcinoma from DES).

normal vaginal bleeding, an abnormal discharge, or post-

per third of the vagina in 75% of patients.

y, and biopsy.

f involved areas when they are few and small.

- Early disease is typically not detected on routine pelvic
- Some 75% of woman present with advanced malign denced by abdominal pain and bloating, a palpable a ascites.
- Table 2.12-11 differentiates the benign and malignan pelvic masses.

### Diagnosis

- **Tumor markers** (see Table 2.12-12): ↑ **CA-125** is ass lial cell cancer (90% of ovarian cancers) but is used progression and recurrence.
  - **Premenopausal women:** ↑ CA-125 may point to as endometriosis.
  - **Postmenopausal women:** ↑ CA-125 (> 35 units) i hood that the ovarian tumor is malignant.
- **Transvaginal ultrasonography:** Used to screen high the first step in the workup of symptomatic women pelvic pain).

**TABLE 2.12-11. Benign vs Malignant Pelvic Masses**

| Finding | benign | |
|---|---|---|
| | **examination: pelvic Mass** | |
| Mobility | Mobile | Fixed |
| Consistency | Cystic | Solid |
| Location | Unilateral | Bilat |
| Cul-de-sac | Smooth | Nodu |

**l women:**

w with ultrasonography asymptomatic, unilateral simple
n diameter with a normal CA-125.
aluate palpable masses.

**n cancer** is as follows:

ng: TAH/BSO with omentectomy and pelvic and paraaor-
nectomy.
asms warrant tumor removal or unilateral oophorectomy.
**hemotherapy:** Routine except for women with early-stage
rian cancer.
**py:** Effective for dysgerminomas.

e BRCA1 gene mutation should be screened annually
aphy and CA-125 testing. Prophylactic oophorectomy is
y age 40 or whenever childbearing is completed.
isk of ovarian cancer.
ne screening for ovarian cancer.

## gan Prolapse

ic organ prolapse include vaginal birth, genetic predispo-
ge, prior pelvic surgery (hysterectomy), connective tissue
ra-abdominal pressure associated with obesity or straining
pation.

e sensation of a bulge or protrusion in the vagina (see Fig-

incontinence, a sense of incomplete bladder emptying,

Dysg

Granu

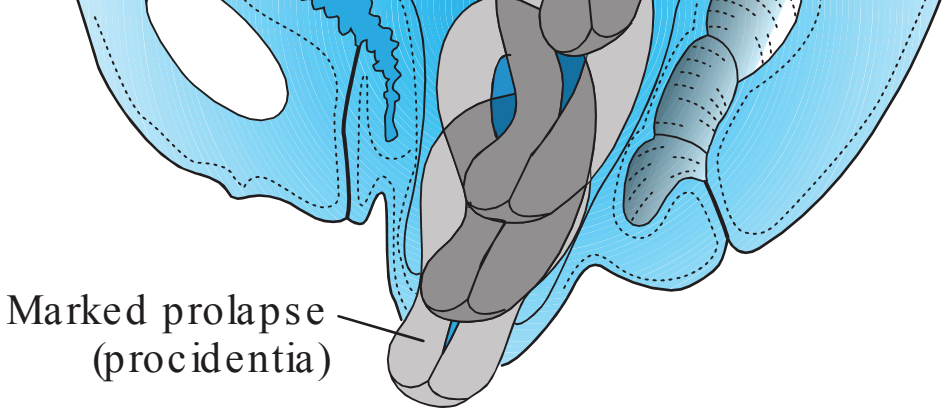



**FIGURE 2.12-11. Uterine prolapse.** Different degrees of uterine p
(Reproduced with permission from DeCherney AH, Nathan L. Current Diagnosis & Treatment C
New York, NY: McGraw-Hill; 2007.)

## Urinary Incontinence

Defined as the involuntary loss of urine due to either b
dysfunction.

### History/Pe

- Table 2.12-13 outlines the types of incontinence alo
guishing features and treatment (see also the mnemon
- Exclude fistula in cases of total incontinence.
- Look for neurologic abnormalities in cases of urge
ticity, flaccidity, rectal sphincter tone) or distended
incontinence.

### Diagnosis/Treatment

- Obtain a UA and urine culture to exclude UTI.
- Voiding diary.
- Consider urodynamic testing.
- Serum creatinine to exclude renal dysfunction.
- Obtain a cystogram to demonstrate fistula sites and de
der neck.

| | | |
|---|---|---|
| ing, sneezing, lifting). | **laxity of pelvic fl or musculature.** Common in multiparous women or after pelvic surgery. | Vaginal |
| , unexpected urge to void that is ed to position or activity. | Detrusor hyperrefl xia or sphincter dysfunction due to inflamm tory conditions or neurogenic disorders of the bladder. | Anticho behavi |
| c urinary retention. | Chronically distended bladder with ↑ intravesical pressure that just exceeds the outlet resistance, allowing a small amount of urine to dribble out. | Placem setti Treat u Timed |

ited contractions, local irritation (cystitis, stone, tumor), and CNS causes.
cal agents (tumor, stricture), neurologic factors (lesions), and medications.

cent antibiotic therapy, immunosuppressed (eg, diabetes).
ren.
ally from sexual abuse.

**vulvovaginitis:** Causes include contact dermatitis and

**ides (rhabdomyosarcoma):** A malignancy with lesions pearance of "bunches of grapes" within the vagina.

RTY

characteristics in a child < 8 years of age. Subtypes are as ble 2.12-14):

CNS trauma/irradiation | Ovarian cysts (females)

Pineal tumors (rare)

Neurofibromatosis with CNS involvement

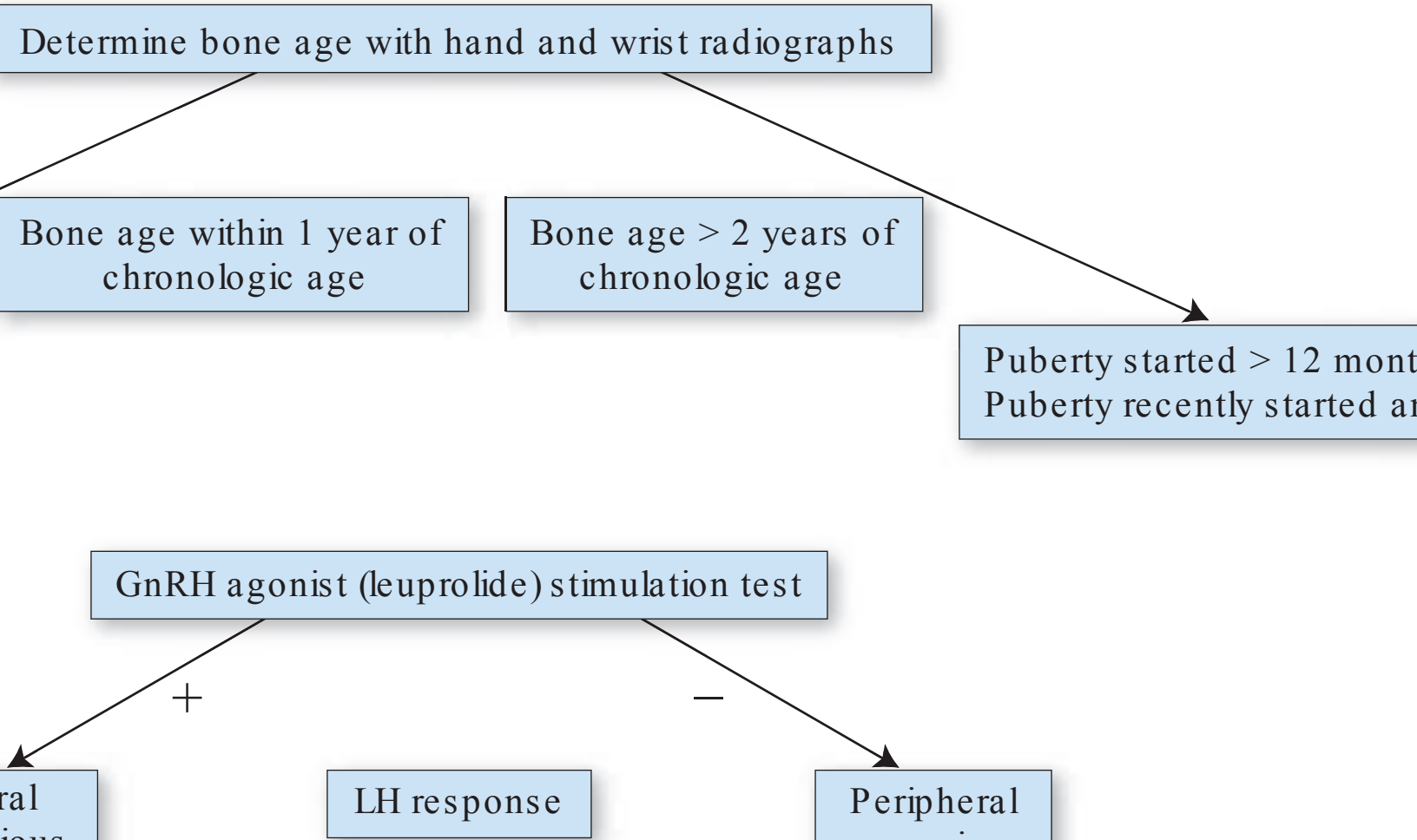
Tuberous sclerosis

## Diagnosis

Figure 2.12-12 illustrates an algorithm for the workup of p

## Treatment

- **Central precocious puberty:** Leuprolide is first-lin changes regress or cease to progress.
- **Peripheral precocious puberty:** Treat the cause.
  - **Ovarian cysts:** No intervention is necessary, as cyst spontaneously.


Determine bone age with hand and wrist radiographs
Bone age within 1 year of chronologic age
Bone age > 2 years of chronologic age
Puberty started > 12 mont
Puberty recently started ar
GnRH agonist (leuprolide) stimulation test
+
–
ral
LH response
Peripheral

sed risk of breast cancer. Simple breast cysts are the most volves fluid-filled masses from an exaggerated stromal tis- nones and growth factors.

e cysts (gross and microscopic), papillomatosis, adenosis, tal epithelial hyperplasia.
women 30–50 years of age; rarely found in postmeno-

rauma and caffeine use.

mastalgia and swelling, most prominent just before men-

n in the size of the masses is common.
include an irregular, bumpy consistency to the breast tis-

13 for an algorithm of a breast mass workup.
nt return after menstruation, because symptoms fluctuate

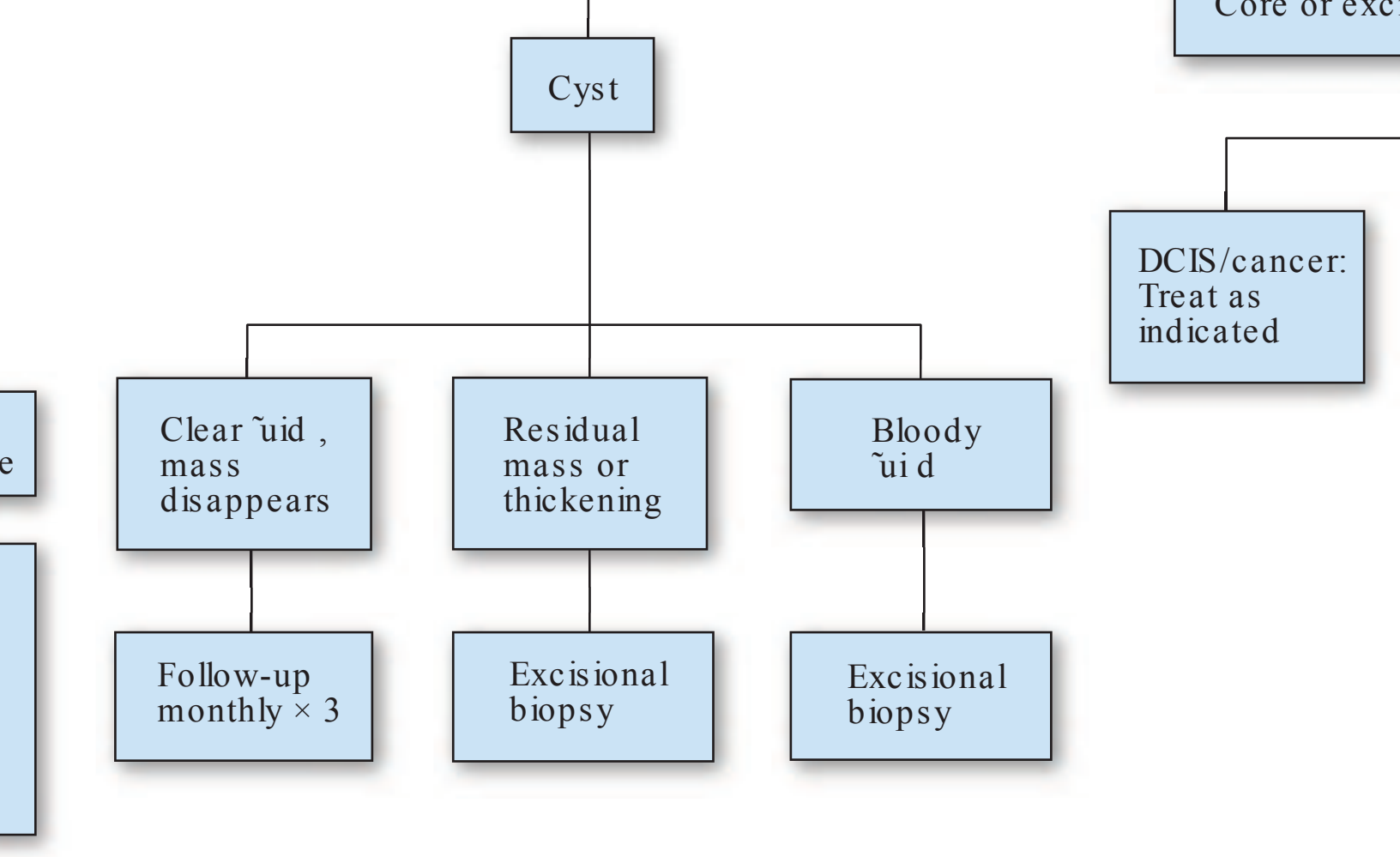
n follow-up, perform ultrasonography to differentiate a illed vs solid.
e aspiration (FNA) of a discrete mass that is suggestive of a to alleviate pain as well as to confirm the cystic nature of

y is indicated if no fluid is obtained or if the fluid is tion.
s of limited use (especially if < 35 years because of density

k of breast cancer if simple cyst, but ↑ risk if complex cyst l hyperplasia or cellular atypia), which is rare.

The diffe
mass inc
fibroade
necrosis

Intraduc
duct ect
bloody




**f a breast mass.**

wall of a cyst into the lumen. It is diagnosed with core ne
gical excision is required since it can be associated with at
rosing adenosis and usual ductal hyperplasia require no tr

## FIBROADENOMA

Proliferative breast lesion without atypia. A benign, slow-g
with epithelial and stromal components. **The most comm
women < 30 years of age.**

### HisTory/Pe

- Presents as round, rubbery, discrete, relatively mobi
  diameter. Generally nontender, but not always.

a (AH) can be ductal or lobular, filling part but not the
e. It is similar to a low-grade DCIS or LCIS with a moder-
f breast cancer. It is a pathologic diagnosis by biopsy often
nogram. AH requires risk reduction, which involves yearly
amoxifen or aromatase inhibitor.

## ancer

cancer (affects one in eight women) and the second most
ancer death in women (after lung cancer) in the United
t **occur in the upper outer quadrant.** Risk factors include
**of newly diagnosed patients have risk factors):**

older age; Caucasian.
ry of breast cancer; family history in a first-degree rela-
ors (BRCA1 and BRCA2 mutations: associated with early

es (controversial).
ation.
cystic change with cellular atypia.
trogen (nulliparity, early menarche, late menopause, first
ncy after age 35).

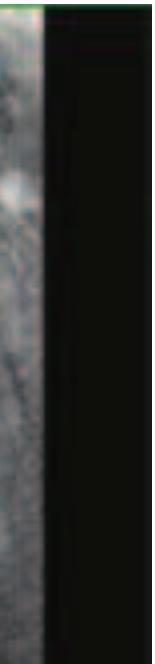
ons include the following:

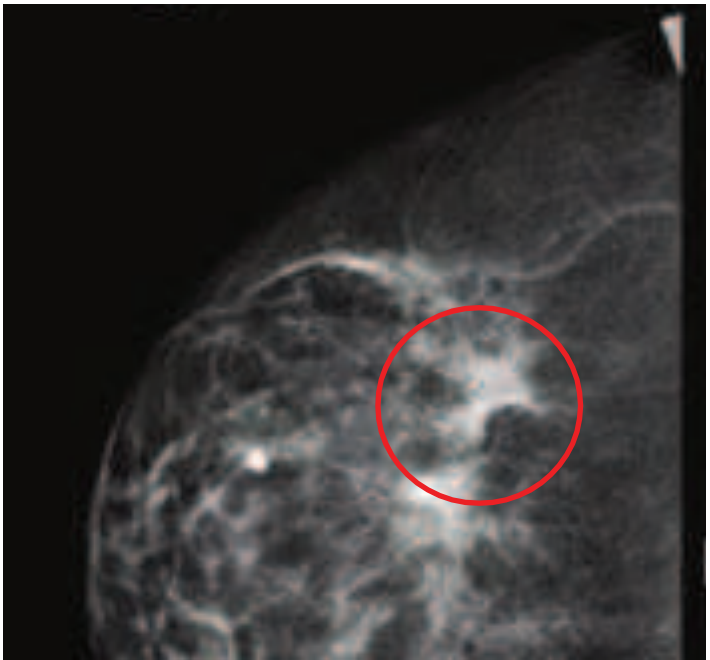
Single, nontender, immovable, firm-to-hard mass with ill-
**OR** mammographic abnormalities on routine screening.
**locally advanced:** Axillary lymphadenopathy, breast
in, peau d'orange skin findings suggesting inflammation

**with cla**
duced with
diate breas
AlloDerm, t
World J Sur

↑ expos
menarch
↑ the ris

- **Biopsy of suspicious lesions on mammography:**
  - Perform a mammography-guided FNA or core thickness skin biopsy if signs of inflammation.
  - **FNA:** A good initial biopsy, especially for lesions cl ever, it is a small sample with a high false-⊕ rate used to follow response to treatment.
  - **Core needle biopsy:** A larger sample that allows status.
  - **Open biopsy:** Less commonly used. Provides tissu rate diagnosis and allows immediate resection requires taking the patient to the OR.
- **Receptor status of tumor:** Determine estrogen recep rone receptor (PR), and human epidermal growth fa status.
- **Tumor markers for recurrent breast cancer:** Include or CA 27-29.
- **Metastatic disease:**
  - **Labs:** ↑ ESR, ↑ alkaline phosphatase (liver and ↑ calcium.
  - **Imaging:** CXR; CT of the chest, abdomen, and PET and bone scans can also be useful.







rapy as below.

**y:**

⊕ patients should receive **tamoxifen** (selective estrogen dulator [SERM] that competitively antagonizes the ER, e growth of breast cancer cells) **OR** aromatase inhibitor if sal (inactivates aromatase that converts peripheral andro-gens).

u-⊕ should receive **trastuzumab,** a monoclonal antibody HER2/neu receptors on the cancer cell.

HER2-⊖ patients should receive **chemotherapy** if tumor **not** give if patient already received neoadjuvant therapy.

**ns to breast-conserving therapy (lumpectomy)** include , subareolar location, multifocal tumors, fixation to the radiation to the chest wall, or involvement of the nipple .

e should be treated with radiation therapy and hormonal omy may be required for local symptom control.

es over the breast cancer stages.

-IV) is the most reliable indicator of prognosis.

status is associated with a favorable prognosis.

l to the breast has a 75–90% cure rate. With spread to the survival rate is 40–50%.

sociated with a poor prognosis.

curs in 50% of patients with metastatic breast cancer; common.

Breast ca
tumor si
with no
associat

A 27-y
cm ×
exami

of the assault (eg, penile penetration, use of condom
use or display of weapons), and the patient's action
(eg, douching, bathing, brushing teeth, urination/d
clothes).

- Conduct a complete physical examination, making
trauma, along with a detailed pelvic examination, in
the external genitals, vagina, cervix, and anus.

### Diagnosis

- Gonorrhea and chlamydia smear/culture (including r
wet mount and culture for trichomonas. On an indivi
serologic testing for HIV, syphilis, HSV, HBV, and CM
- Serum pregnancy test.
- Blood alcohol level; urine toxicology screen.

### Treatment

- Treat traumatic injuries.
- STD treatment/prophylaxis (ceftriaxone plus azithron
zole); Hep B vaccination if patient has not received ful
- HIV risk assessment and possible postexposure prophyl
- Emergency contraception for pregnancy prevention.
- Refer for psychological counseling.
- Arrange for follow-up with the same physician or with
more appropriate.
- Follow-up should include repeat screening for STDs,
pregnancy, and a discussion of coping methods with
for psychiatric care if needed.

A

< 30
y is
ishing

ase

US

TA

EAT VESSELS

NES

logical Disease

ULUS

IS

liated Disease

d ERS

RITIS

ease

370 PERTUSSIS (WHOOPING COUGH)

371 VIRAL EXANTHEMS

372 TORCHES INFECTIONS

373 **Neonatology**

375 APGAR SCORING

376 CONGENITAL MALFORMATIONS

377 NEONATAL JAUNDICE

378 RESPIRATORY DISTRESS SYNDROME

378 **Pediatric Neurologic Disease**

380 CEREBRAL PALSY

380 FEBRILE SEIZURES

381 **Pediatric Oncology**

384 LEUKEMIA

385 NEUROBLASTOMA

385 WILMS TUMOR

386 CHILDHOOD BONE TUMORS

387 **Pediatric Musculoskeletal Disord**

387 COMMON PEDIATRIC ORTHOPEDIC INJUR

388 DUCHENNE MUSCULAR DYSTROPHY

388 DEVELOPMENTAL DYSPLASIA OF THE HIP

389 LEGG-CALVÉ-PERTHES DISEASE

389 SLIPPED CAPITAL FEMORAL EPIPHYSIS

392 SCOLIOSIS

394 **Well-Child Care**

394 ANTICIPATORY GUIDANCE

394 HEARING AND VISION SCREENING

- Suspect abuse if the story is not consistent with the in the child's developmental age. For example, take note that their 2-month-old child "rolled off the couch" (2-m yet). Also suspect abuse if the story continually changes
- **Risk factors:** Look for parents with a history of alcol children with complex medical problems, and repeated
- **Infants:** Abuse or neglect in infants may present as ap ing intolerance, excessive irritability, somnolence, (FTT).
- **Older children:** Neglect in older children may presen behavioral abnormalities.

**See** Table 2.13-1 **for examination findings.**

Diagnosis

- An **x-ray skeletal survey** and **bone scan** can show stages of healing. Radiographs may not show fractur after injury (although they may show evidence of prior

**TABLE 2.13-1. Common Presentations and Mimics of Child Abus**

| Type of Abuse | Presentation/Imaging Findings | |
|---|---|---|
| Bruises | Most common physical findin . Often located on head and torso. May be in pattern refle ting implement (hand, belt). | Mongolian s Coining/cup treatments |
| Burns | Contact burns: cigarette/curling iron. Immersion burns: hot water, appear on buttocks or stocking-glove distribution. | Scalded skin contact derm |



**ongolian spots.** These spots, which are common and are not patho-
s of life, often resemble bruises. (Reproduced with permission from Wolff Ket al.
ral Medicine, 7th ed. New York, NY: McGraw-Hill; 2008.)

); by contrast, bone scans may show fractures within 48

s suspected, test for gonorrhea, syphilis, chlamydia, HIV,
n 72 hours of assault).
e head trauma (formerly referred to as shaken baby syn-
rming an ophthalmologic exam for **retinal hemorrhages**
**st CT** for subdural hematomas. Infants with abusive head
not exhibit external signs of abuse.
**RI** to visualize white-matter changes associated with vio-
the extent of intra- and extracranial bleeds.

ies, including location, size, shape, color, and the nature
ises, or burns.
tective Services for possible removal of the child from the

cessary to stabilize injuries or to protect the child.

## tal Heart Disease

Neisseria
vaginal
sexual a
not, bec
the mot
persist f

Cyanot

**The 5 t**
**to-left**

**T**runcus
overri
**T**ranspos
**2** arte
**Tri**cuspi
**Tetra**log
**T**otal an
= **5** w

Out of th
presents
first ew

| | |
|---|---|
| Coronary artery aneurysms | Kawasaki disease. |
| Congenital heart block | Neonatal lupus. |
| Supravalvular aortic stenosis | Williams syndrome. |
| Conotruncal abnormalities | Tetralogy of Fallot (ove arteriosus, DiGeorge s velocardiofacial syndr |
| Ebstein abnormality (apical displacement of the tricuspid valve leading to atrialization of the RV) | Maternal lithium use d |
| Heart failure | Neonatal thyrotoxicos |
| Asymmetric septal hypertrophy and transposition of the great vessels | Maternal diabetes. |

## SEPTAL DEFECTS

A condition in which an opening in the atrial or ventric blood to flow between the atria or ventricles, leading to le **Ventricular septal defect (VSD) is the most common heart disease.**

### Presentation and Diagnosis

See Table 2.13-3.

### Treatment

| | | |
|---|---|---|
| | **Small defects:** Asymptomatic.<br>**Large defects:** Easy fatigability; frequent respiratory infection; FTT. | **Small defects:** Asyn<br>**Large defects:** Recu dyspnea; FTT; CHI |
| | Wide and fi ed split S2.<br>Systolic ejection murmur at the left USB (↑ fl w across pulmonary valve).<br>Mid-diastolic rumble at the left LSB. | Harsh holosystolic n defects).<br>Narrow S2 with ↑ P2<br>Mid-diastolic apical across mitral valve |
| | Cardiomegaly.<br>↑ pulmonary vascular markings. | |
| | RVH.<br>Right atrial enlargement.<br>PR prolongation is common. | **LVH** (RVH may also |
| ng | Defect and blood fl w across the atrial/ventricular septum. | |

## RTERIOSUS

s arteriosus to close in the first few days of life, leading to right shunt from the aorta to the pulmonary artery (Figure

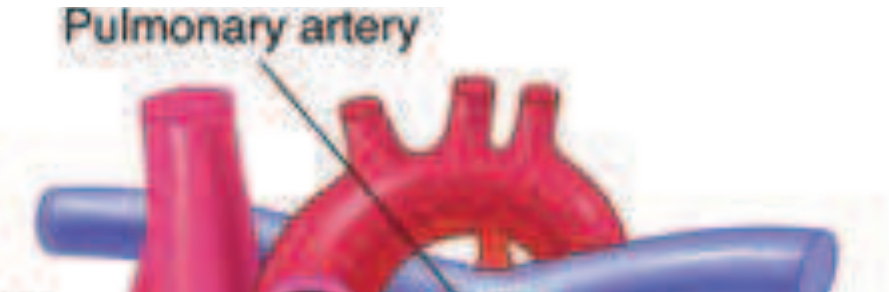





**FIGURE 2.13-3. Ventricular septal defect with resultant left-to-r**
with permission from USMLE-Rx.com.)

ke state
look

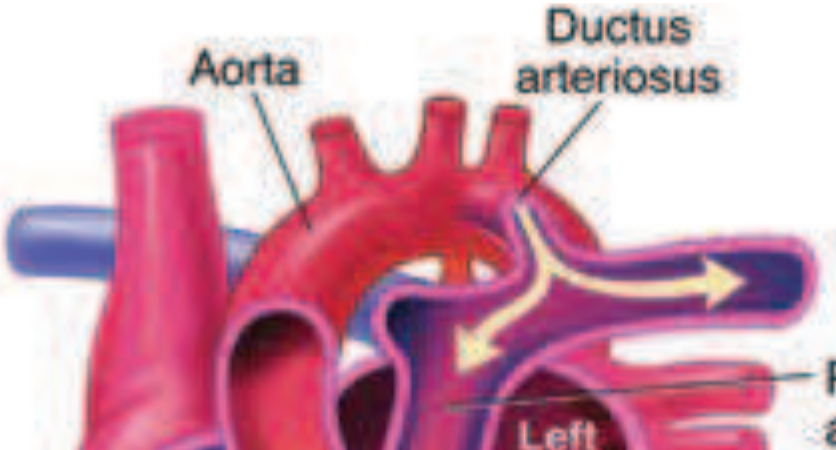
n.
ital
ided
sing).
lasia.

## History/Pe

- Typically asymptomatic; patients with large defects ma
  recurrent lower respiratory tract infections, clubbing, a
- Examination reveals a **continuous "machinery mur**
  left intercostal space at the sternal border, a loud S2, **w**
  and **bounding peripheral pulses.**

## Diagnosis

- **Echocardiogram** is the diagnostic test of choice: Colo
  demonstrate blood flow from the aorta into the pulmon
- With larger PDAs, echocardiography shows left atrial
  enlargement.
- ECG may show LVH, and CXR may reveal cardion
  large.

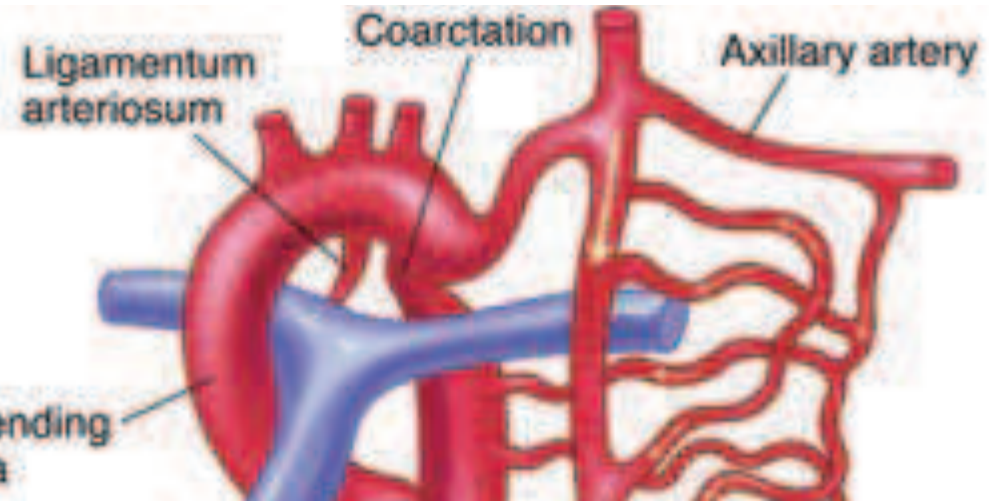


ortion of the aorta, leading to ↑ flow proximal to and coarctation (Figure 2.13-5). Occurs just distal to the left 98% of patients. The condition is associated with **Turner** neurysms, and male gender. **More than two-thirds of uspid aortic valve.**

Coarctat
with Tur

in childhood with **asymptomatic hypertension (upper tension);** the classic physical examination finding is a is **higher in the upper extremities;** the difference in BP and right arm can indicate the point of coarctation.
urmur may be heard diffusely across the torso if well-terals are present.
claudication, syncope, epistaxis, and headache may be

ngs include **weak femoral pulses,** radiofemoral delay, a rmur in the left axilla, and a forceful apical impulse.
cal coarctation requires a patent PDA for survival. Such ent in the first few weeks of life with poor feeding, leth- and eventual shocklike state when the PDA closes. Dif- s may be seen with lower $O_2$ saturation in the left arm and s (postductal areas) as compared to the right arm (preduc-



## Treatment

- If severe coarctation presents in infancy, the ductus a
  kept open with prostaglandin $E_1$ ($PGE_1$).
- Surgical correction or balloon angioplasty is controvers
- Monitor for restenosis, aneurysm development, and ao

## TRANSPOSITION OF THE GREAT VESSELS

**The most common cyanotic congenital heart lesion in**
ure 2.13-7**).** In this condition, the aorta is connected to th
the pulmonary artery to the left ventricle, creating para
systemic circulations. **Without a septal defect (ASD or**
**it is incompatible with life.** A PDA alone is usually no
adequate mixing of blood. Risk factors include **diabetic**
DiGeorge syndrome.

n)

hiltrum,

oplasia/

### History/Pe

- Critical illness and **cyanosis typically present within**
  **birth.** Reverse differential cyanosis may be seen if left
  tract obstruction (eg, coarctation, aortic stenosis) is also
- Examination reveals tachypnea, progressive hypoxemi








**mplete transposition of the great vessels.** The right ventricle deoxy-
l to the aorta and oxygenated blood is directed back to the pulmonary
nission from USMLE-Rx.com.)

ents have signs of CHF, and a single loud S2 is often pres-
not be a murmur if no VSD is present. If a VSD is pres-
rmur may be heard at the left sternal border.

ny.
a narrow heart base, absence of the main pulmonary
an **"egg-shaped silhouette,"** and ↑ pulmonary vascular

maintain or open the PDA.
feasible within the first few days of life or if the PDA can-
ed with prostaglandin, perform **balloon atrial septostomy**
rge an ASD.
on (arterial or atrial switch) is definitive.

LOT

flow tract obstruction, overriding aorta, RVH, and VSD
**e most common cyanotic congenital heart disease in**
nosis results from right-to-left shunting across the VSD. As

Transpo
most co
of **newb**
most co
of **child**




**FIGURE 2.13-8. Tetralogy of Fallot.** Substantial right ventricular
resulting in right-to-left shunting through the ventricular septal defect.
from USMLE-Rx.com.)

- **Children often squat** for relief during hypoxemic
  **spells,"** which ↑ systemic vascular resistance, thus in
  to the pulmonary vasculature and improved oxygenatio
- Hypoxemia may lead to FTT or mental status changes.
- Examination reveals a systolic ejection murmur at th
  border (right ventricular outflow obstruction), a righ
  and a single S2.

### Diagnosis

- Echocardiography and catheterization.
- CXR shows a **"boot-shaped" heart** with ↓ pulmonary
  Remember that a VSD may result in ↑ pulmonary vasc
- ECG shows right-axis deviation and RVH.

### Treatment

- Lesions with severe RV outflow tract obstruction or at
  diate $PGE_1$ to keep the PDA open along with urgent su
- Treat hypercyanotic "tet spells" with $O_2$, propranolol,
  knee-chest position, fluids, and morphine.
- Temporary palliation can be achieved through the cre
  shunt (eg, balloon atrial septostomy) before definitive
  (Blalock-Taussig shunt).

| | | |
|---|---|---|
| **unassisted.** | Transfers objects; demonstrates raking grasp. | **Babbles.** |
| vls; cruises; pulls to d. | Uses **3-finger (imm ture) pincer grasp.** | Says "mama/dada" (nonspecific); s ys first orc at 11 months. |
| **ks alone;** throws object. | Uses **2-finger (m ture) pincer grasp.** | Uses **1–3 words.** |
| **ks up/down steps;** ps. | Builds tower of 6 cubes. | Uses **2-word phrases.** |
| **es tricycle;** climbs stairs alternating feet (3–4 s). | **Copies a circle;** uses utensils. | Uses **3-word sentences.** |
| s. | **Copies a cross** (square at 4.5 years). | Knows colors and some numbers. |
| s; walks backward for distances. | **Copies a triangle;** ties shoelaces; knows left and right; prints letters. | Uses **5-word sentences.** |

2 years of age, chronologic age must be adjusted for gestational age. For example, an i
e expected to perform at the 4-month level at the chronologic age of 6 months.

**or Milestones in Language Development**

Infants can be expected to **double their BW by 4–5 year, and quadruple by 2 years.**

- **FTT:** Persistent weight less than the fifth percentile fo the growth curve" (ie, crossing two major percentile chart). Classified as follows:
  - **Organic:** Due to an underlying medical condition sis, congenital heart disease, celiac sprue, pylor infection (eg, HIV), and GERD.
  - **Nonorganic:** Primarily due to psychosocial facto inaccurate mixing of formula (too much water r depression, neglect, or abuse.
- A careful dietary history and close observation of mat tions (especially preparation of formula and feeding) a sis.
- Children should be hospitalized if there is evidence malnourishment. Calorie counts and supplemental feeding is inadequate) are mainstays of treatment.

## SEXUAL DEVELOPMENT

- **Tanner staging:** Performed to assess sexual developme (see Figure 2.13-9). Stage 1 is preadolescent; stage 5 stages are assigned for testicular and penile growth growth in girls; pubic hair development is used for both
  - **Girls:** The average age of puberty is **10.5 years.** menarche in US girls is 12.5 years.
  - **Boys:** The average age of puberty is **11.5 years.**

Normal sexual development:

→ pubarche → growth spurt → m

**uberty delay:** Rarely, due to systemic disease (eg, IBD), (eg, anorexia nervosa), gonadal dysgenesis (eg, Klinefelter urner syndrome), or endocrine abnormalities (eg, hypopi- bothyroidism, Kallmann syndrome, androgen insensitivity ader-Willi syndrome).



Trisomi

21—Age
18—Age
syndr
13—Age

## Disease

8 outline common genetic diseases and their associated

**omies**

| genetic abnormality | common characteris |
|---|---|
| Trisomy 21 as a result of meiotic nondisjunction (95%), Robertsonian translocation (4%), or mosaicism (1%). | The most common chromosomal disorder and caus<br>Associated with **advanced maternal age.**<br>Presents with intellectual disabilities, a fl t facial pr<br>epicanthal folds, **single palmar crease, general**<br>(nuchal folds are sometimes seen on prenatal ult<br>Associated with **atlantoaxial instability, duodena**<br>**disease,** and **congenital heart disease.** The mo<br>malformation is AV canal (60%); ASDs, VSDs, and<br>congenital heart disease make up the remainder<br>Also associated with an ↑ risk of **ALL, hypothyroid**<br>**Alzheimer disease.** |
| Trisomy 18. | Presents with severe intellectual disabilities, **rocker**<br>**micrognathia, clenched hands (overlapping fo**<br>prominent occiput |

| | | |
|---|---|---|
| | | **tall stature,** long extremities, **gyn**<br>**hair distribution.**<br>Treat with **testosterone** (prevents gy<br>2° sexual characteristics). |
| Turner syndrome (female) | 45,XO | Missing 1 X chromosome; no Barr bo<br>Not associated with advanced mater<br>The most common cause of **1° amen**<br>**dysgenesis** (treat with estrogen).<br>Features include short stature, shield<br>nipples, a **webbed neck, coarctat**<br>(↓ femoral pulses), and/or bicuspid<br>present with **lymphedema of the**<br>**neonatal period.** May have horses |
| Double Y males | 47,XYY | Observed with ↑ frequency among in<br>institutions.<br>**Often look normal;** some patients a<br>acne (seen in 1–2% of XYY male pa |

**ic Diseases**

| Etiology | Mode o |
|---|---|
| by ↓ phenylalanine hydroxylase or ↓ tetrahydrobiopterin cofactor.<br>e becomes essential, and phenylalanine accumulates and is<br>equently converted to its ketone metabolites.<br>l at birth; presents within the first few months of life.<br>ts with intellectual disabilities, **fair hair and skin, eczema, blond hair,**<br>**eyes, and a musty urine odor.** | Autosomal re<br>PKU is screen |

| | |
|---|---|
| Findings include **renal failure and an ↑ risk of stroke and MI (thromboembolic events).** | |
| Absence of galactosylceramide and galactoside (due to galactosylceramidase deficien y), leading to the accumulation of galactocerebroside in the brain.<br>Characterized by **progressive CNS degeneration,** optic atrophy, spasticity, and death within the first 3 ears of life. | Aut |
| Caused by a deficien y of **glucocerebrosidase** (also known as acid β-glucosidase) that leads to the accumulation of glucocerebroside in the brain, liver, spleen, and bone marrow.<br>Gaucher cells have a characteristic **"crinkled paper"** appearance with enlarged cytoplasm.<br>May present with **anemia and thrombocytopenia.**<br>The infantile form results in early, rapid neurologic decline. The adult form (more common) is compatible with a **normal life span** and does not af ect the brain. | Aut |
| A deficien y of **sphingomyelinase** that leads to the buildup of sphingomyelin cholesterol in reticuloendothelial and parenchymal cells and tissues.<br>Patients with type A die by age 3.<br>May present with a **cherry-red spot and hepatosplenomegaly.** | Aut<br>No<br>n |
| An absence of hexosaminidase that leads to $GM_2$ ganglioside accumulation.<br>Infants may appear **normal until 3–6 months of age,** when weakness begins and development slows and regresses. An exaggerated startle response may be seen.<br>Death occurs by age 3.<br>Presents with a **cherry-red spot but no hepatosplenomegaly.** | Aut<br>**Tay** |

- Neonates most commonly present with obstruction due to abnormally thick meconium (15% of presen
- Patients < 1 year present with cough, wheezing, c tory infections. They may also have steatorrhea and
- Most patients > 1 year present with **FTT (due to ciency)** or **chronic sinopulmonary disease/sputum**

- Affected individuals exhibit **recurrent pulmonary in** with Pseudomonas and Staphylococcus aureus) with s **digital clubbing, chronic cough** (the most commo tom), dyspnea, bronchiectasis, hemoptysis, chronic rales, hyperresonance to percussion, and **nasal polypos**
- Patients with pancreatic insufficiency usually have **gr** ulence; other prominent GI symptoms include panc **lapse,** hypoproteinemia, biliary cirrhosis, jaundice, a ces.
- Patients who present in late childhood or adulthood ar creatic sufficiency; in this case, pulmonary manifest and the disease tends to have a milder course.
- Additional symptoms include **type 2 DM, "salty-tastin tility** (agenesis of the vas deferens), and **unexplained h**
- Patients are at risk for **fat-soluble vitamin deficiency** and K) 2° to malabsorption and may present with ma deficiencies (ie, night blindness, rickets, neuropathy, c

eus are

## Diagnosis

- Diagnosed by a **sweat chloride test** (pilocarpine electro
- Confirmed by genetic testing.
- Most states now perform **mandatory newborn screenin**

## Treatment

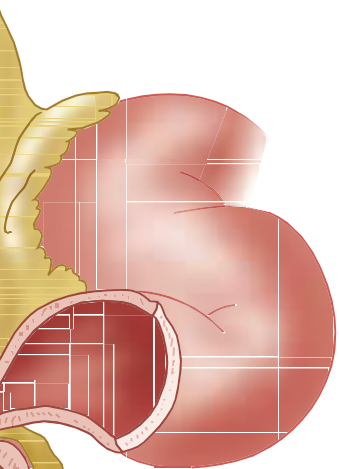
- Pulmonary manifestations are managed with chest phy chodilators, corticosteroids, antibiotics **(should cover** DNase.

**culum,** intestinal lymphoma (> 6 years of age), submuco- Henoch-Schönlein purpura), polyps, and CF (lead point Antecedent GI or URI is seen in many children, which n of a lead point through enlargement of Peyer patches.

abrupt-onset, **episodic abdominal pain** in apparently , often accompanied by flexed knees and vomiting. The r well between episodes if intussusception is released.
l is **severe abdominal pain, vomiting (initially nonbil- us as obstruction develops), and bloody mucus in stool tool,"** a late finding).
, look for abdominal tenderness, a ⊕ stool guaiac, a pal- **shaped" RUQ abdominal mass,** and "empty" RLQ on e sign).

The clas
vomiting
occurs i

y is the test of choice and may show a **"target sign"** (see ); ultrasound must be carried out **during a painful epi-** diagnose intussusception.
**n films** are often normal early in the disease, but later mall bowel obstruction, perforation, or a soft tissue mass.





Hypertrophy of the pyloric sphincter, leading to gastric
More common in **firstborn infant boys;** associated with
fistula, formula feeding, and **maternal erythromycin** inges

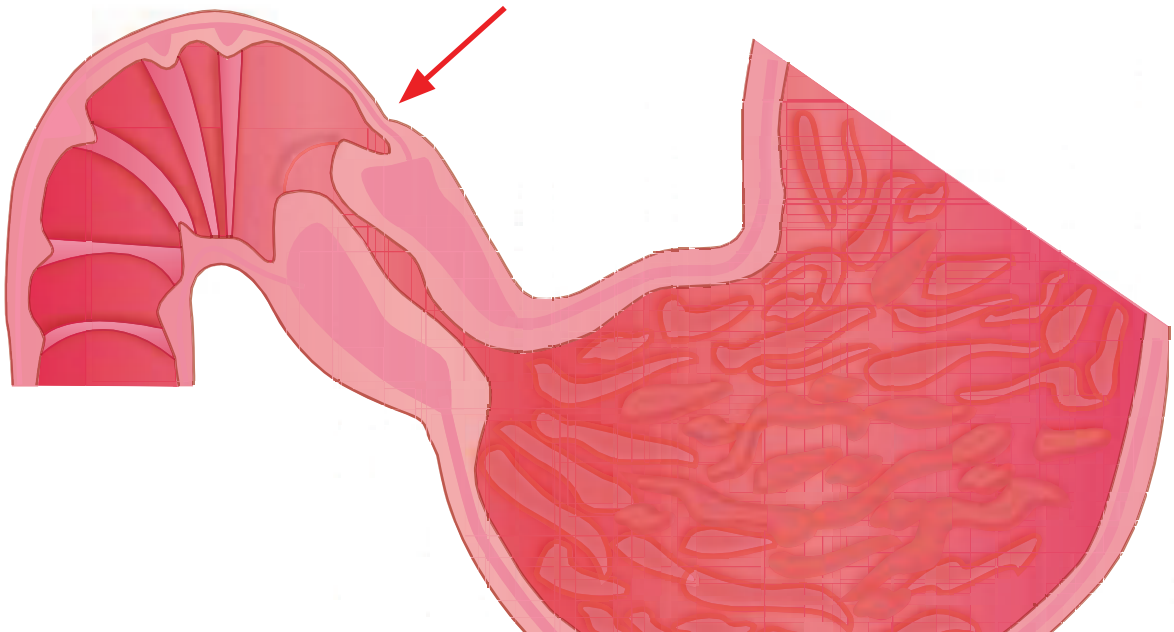
### History/Pe

- **Nonbilious emesis** typically begins around **3–5 wee**
  gresses to **projectile emesis** after most or all feedings.
- Infants are generally hungry after episodes of vomitin
  well but eventually suffer from dehydration and malnu
- Examination may reveal a **palpable olive-shaped,** mo
  gastric mass and visible gastric peristaltic waves.

### Diagnosis

- **Abdominal ultrasound** is the imaging modality of c
  thickened, elongated pylorus (see Figure 2.13-11).
- **Laboratory workup reveals hypochloremic, hypokale**
  **losis; emesis results in loss of HCl and renin-angi**
  **axis activation.**
- Barium studies show a narrow pyloric channel ("strin
  beak.

mon
nfancy;
etween





testinal ulceration and painless hematochezia. This is the genital abnormality of the small intestine, affecting up to ys > girls). Most frequently symptomatic in **children < 2**

tomatic, and often discovered incidentally.
nts with **painless rectal bleeding.**
include intestinal perforation or obstruction, diverticulitis ic acute appendicitis), and intussusception.

**graphy scan** (technetium-99m pertechnetate; detects ec- e) is diagnostic.
limited value but can be useful in diagnosing obstruction

**al excision** of the diverticulum together with the adjacent h may be ulcerated. Indications for urgent/emergent sur- rrhage, diverticulitis, intestinal perforation, and obstruc- .

SEASE

ganglion cells in the distal colon, leading to decreased opposed smooth muscle tone in the absence of enteric d uncoordinated peristalsis (Figure 2.13-12). Associated **Down syndrome,** Waardenburg syndrome, and multiple type 2.

Bleeding
complic
may be
cause h



**FIGUR**
**disease.**
shows sn
and rect
dilated t
in the de
sent fece
USMLE-Rx.

rectum.

## MALROTATION WITH VOLVULUS

Congenital malrotation of the midgut results in abnorma
small intestine (cecum in the right hypochondrium) and
bands (Ladd bands), which predispose to obstruction
blood flow.

### History/Pe

- Often presents in the **first month of life** with **bili**
abdominal pain, distention, and passage of blood or mu
- Postsurgical adhesions can lead to obstruction and volv
life.

### Diagnosis

- AXR may reveal the characteristic **"bird-beak" appe**
levels but may also appear normal.
- **Upper GI series** is the study of choice if the patient is
abnormal location of the ligament of Treitz. Ultrasoun
its sensitivity is contingent on the experience of the ult

### Treatment

- NG tube insertion to decompress the intestine; IV fluid
- Emergent surgical repair when volvulus is gastric; su
when volvulus is intestinal.

## NECROTIZING ENTEROCOLITIS

A condition in which a portion of the bowel (most com
ileum/proximal colon) undergoes necrosis. The **most**

also be helpful in discerning free air, areas of loculation, sis.

ve measures, including NPO, an orogastric tube for gastric correction of dehydration and electrolyte abnormalities, ibiotics.

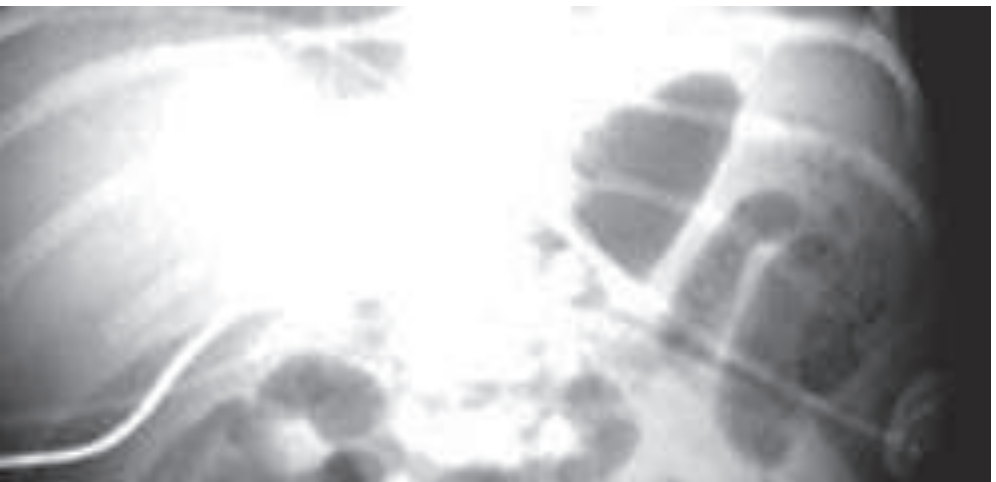
**surgery are perforation (free air under the diaphragm) diographic signs** on serial abdominal plain films. An ile- cous fistula is typically performed, with a reanastomosis

nclude formation of intestinal **strictures** and **short-bowel**

## Immune-Mediated Disease

### CY DISORDERS

odeficiencies are rare and often present with chronic or (eg, chronic thrush), unusual or opportunistic organisms, nt response, or FTT. Categorization is based on the single nponent that is abnormal (see also Table 2.13-9).



Flashbac

- **B ce** and agai
- **T ce** agai **viru**

| | | |
|---|---|---|
| nger active. | | provide a<br>Treat with p<br>and **IVIG.** |
| lly a combined B- and T-cell<br>fect.<br>**g levels are low** (in the 20s and<br>s).<br>**nal B-cell numbers; ↓ plasma**<br>**lls.**<br>ptoms usually present later in<br>(15–35 years of age). | ↑ pyogenic upper and lower respiratory infections.<br>↑ risk of lymphoma and autoimmune disease. | Quantitative<br>B- and T-c<br>**Treat with** |
| **the most common**<br>**mmunodeficien y.**<br>**A levels only.** | Usually asymptomatic; patients may develop recurrent respiratory or GI infections (Giardia).<br>**Anaphylactic transfusion reaction** due to anti-IgA antibodies is a common presentation. | Quantitative<br>infections<br>**Be careful g**<br>can lead t<br>anti-IgA a<br>severe all<br>is necessa<br>IVIG. |

## t-cell disorders

| | | |
|---|---|---|
| he mnemonic **CATCH 22.**<br>ents with **tetany** (2° to<br>**pocalcemia**) in the first d ys<br>life.<br>somal dominant. | Variable risk of infection.<br>↑↑↑ infections with **viruses, fungi, and PCP pneumonia.**<br>X-ray film m y show **absent thymic shadow.** | Absolute T-<br>mitogen<br>delayed h<br>testing.<br>Treat with b<br>transplan |

Referred to as "bubble boy disease," because children are confined to an isolated, sterile environment.

An **X-linked recessive** disorder seen **only in male patients.**

Symptoms usually present at birth.

Patients have ↑ IgE/IgA, ↓ **IgM,** and **thrombocytopenia.**

The classic presentation involves **bleeding, eczema, and recurrent otitis media.**

Remember the mnemonic **WIPE:**

**W**iskott-Aldrich **I**nfections

**P**urpura (thrombocytopenic)

**E**czema.

↑↑ risk of atopic disorders, lymphoma/leukemia, and infection from S pneumoniae, S aureus, and H influenzae type b (**encapsulated organisms;** think back to how IgM functions).

Tre
a
**Pa**
a
Pat
t

**cies:** Most common (50%). Typically present **after 6** with recurrent sinopulmonary, GI, and urinary tract infec- **psulated organisms** (Haemophilus in*fl*uenzae, Streptococ- Neisseria meningitidis). **Treat with IVIG (except for IgA**

maglobulinemia can be confused with transient hypo- linemia of infancy (THI), as both are characterized by ty to infections at ~ 6 months of age, when transplacen- IgG is no longer active. B cells are ↓ in Bruton, whereas are normal.

mmaglobulinemia and common variable immunodefi- D) also have similar symptoms, but the former is found months of age, whereas CVID is seen in older men and

| | | |
|---|---|---|
| e present. | Candida, Klebsiella, Pseudomonas, Aspergillus).<br>May have granulomas of the skin and GI/GU tracts. | **Treat with** judicious during inf<br>IFN-γ can ↓ t infection.<br>BMT and ge therapies. |
| efect in the chemotaxis of eukocytes.<br>nagocytic activity. | Recurrent skin, mucosal, and pulmonary infections. May present as **omphalitis in the newborn** period with **delayed separation of the umbilical cord (> 14 days postbirth).** | **No pus with inflamm** to a chem<br>High WBCs i<br>BMT is curat |
| autosomal recessive disorder hat leads to a defect in eutrophil chemotaxis/ icrotubule polymerization. syndrome includes **partial culocutaneous albinism, eripheral neuropathy, and eutropenia.** | ↑↑ incidence of overwhelming pyogenic infections with Streptococcus pyogenes, S aureus, and Pseudomonas species. | Look for gia neutrophi<br>BMT is the t |
| efect in neutrophil chemotaxis.<br>nember the mnemonic **FATED:**<br>rse **F**acies<br>cesses (S aureus)<br>ained primary **T**eeth<br>er-Ig**E** (eosinophilia)<br>matologic (severe eczema) | Recurrent S aureus infections and abscesses. | Treat with p antibiotics a |

**meningococcal or gonococcal.**
Rarely, lupus or glomerulonephritis.

**:** 2–8 weeks from onset. Manifestations are thrombocyto- ESR. Untreated children may begin to develop **coronary s** (40%); all patients should be assessed by **echocardiogra-**

**> 8 weeks from onset;** begins when all clinical symptoms d, and lasts until ESR returns to baseline. **Untreated chil- of aneurysmal expansion and MI.**

up:
Normochromic anemia, leukocytosis with left shift, high P.
**ase:** Thrombocytosis. ESR and CRP gradually decrease hase.
**n** at time of diagnosis to establish a baseline for longitu- of coronary artery morphology. Follow-up for uncompli- ly at 2 weeks and 6–8 weeks after diagnosis.

(for antiinflammatory, antipyretic, and antithrombotic **G** (to prevent coronary artery aneurysms).

**nical Manifestations of Kawasaki Disease**

| **t least 4 of the following 5 criteria:** |
|---|

Untreate
to coron
patients



**FIGURE ease.** St
swollen
Dong Soo

er may
ngue,"
s and
us
with
d no

and gradual loss of motion that is present for at least 6 wee
**years of age.** Formerly known as juvenile rheumatoid arth
mately 95% of cases resolve by puberty. More common i
The subtypes of JIA are described in Table 2.13-13.

### Diagnosis

See Table 2.13-13.

### Treatment

- **NSAIDs** and strengthening exercises.
- Corticosteroids (for carditis) and immunosuppressive r
  trexate, anti–tumor necrosis factor agents such as etar
  line agents.

# Pediatric Infectious Disease

## ACUTE OTITIS MEDIA

A suppurative infection of the middle ear cavity that is co
Up to 75% of children have at least 3 episodes by age
gens include S pneumoniae, **nontypeable** H in*fl*uenzae,
viruses such as influenza A, RSV, and parainfluenza virus.

**iopathic Arthritis Subtypes**

| **Presentation** | **RF and ANA Status** | |
|---|---|---|
| or fewer joints (usually<br>pearing); no systemic<br>ns. | ANA ⊕<br>RF ⊖ | **Most common**<br>Usually diagnc |

**xicillin** (80–90 mg/kg/day) × 10 days for empiric therapy.
ay require amoxicillin/clavulanic acid.
nclude TM perforation, mastoiditis, meningitis, cholestea-
ic otitis media. Recurrent otitis media can cause hearing
nt speech and language delay. Chronic otitis media may
stomy tubes.

tory illness of the small airways of the lower respiratory
affects infants and children **< 2 years of age,** often in the
**V is the most common cause;** others include parainflu-
etapneumovirus, and other viruses. Progression to respi-
otentially fatal complication. Risk factors for severe RSV
ge < 6 months, prematurity, heart or lung disease, neuro-
nd immunodeficiency.

ow-grade fever, rhinorrhea, cough. Young infants might

spiratory distress, tachypnea, hypoxia.
veals **tachypnea, hypoxia,** intercostal retractions, **crackles**
**sounds ("washing machine sounds"),** ± **wheezing.**
**rate** is the earliest and most sensitive vital sign change.

clinical diagnosis; routine cases do not need any lab or
p.
a CXR can be obtained to rule out pneumonia and may
tion of the lungs with flattened diaphragms, interstitial
electasis.

bronchi
commo

Young i
result of

A4-da
for int
has ex
vomiti
and ha
bright
initiall
only) b
Exami
abdon
bowel
likely d
you ex
radiog

A2-ye
pediat
infecti

An acute viral inflammatory disease of the larynx, prima
glottic space. Pathogens include **parainfluenza virus type**
**2,** and **3** as well as RSV, influenza, and adenovirus. Bact
may progress to tracheitis.

## History/Pe

Prodromal URI symptoms are typically followed by low-g
pnea, **inspiratory stridor** that worsens with agitation, a
characteristic **barking cough** (which worsens at night).

A

tizing
tion
mula
sis
gs on
atosis
ous

## Diagnosis

- Diagnosed by clinical impression; often based on the c
respiratory distress.
- AP neck film may show the **classic "steeple sign" fi**
**rowing** (see Figure 2.13-15), but this finding is **neithe**
**cific.**
- Table 2.13-14 differentiates croup from epiglottitis and

A

k-
omal
s/

## Treatment

- **Mild cases:** Outpatient management with cool mist th
- **Moderate cases:** May require supplemental $O_2$, ora
**roids,** and **nebulized racemic epinephrine.**



**ute-onset high fever** (39–40°C [102–104°F]), **dysphagia,** fled voice, **inspiratory retractions,** cyanosis, and soft stri-

the **neck hyperextended and the chin protruding** ("sniff- n) and **lean forward in a "tripod" position** to maximize

ion can rapidly lead to life-threatening airway obstruction rrest.

nical impression. The differential diagnosis must include ized causes of airway obstruction (see Tables 2.13-14 and

**t be secured before a definitive diagnosis can be made.** tial laryngospasm and airway compromise, **do not exam- nless an anesthesiologist or otolaryngologist is present.** osis is made via direct fiberoptic visualization of a cherry- glottis and arytenoids.
m shows a swollen epiglottis obliterating the valleculae **gn";** see Figure 2.13-16).

Epiglott
can lead
obstruct

**true emergency,** so time should not be wasted on order-

ABCs; **secure the airway first** with endotracheal intuba- omy, and then give IV antibiotics (ceftriaxone or cefurox-

| | | |
|---|---|---|
| odrome (1–7 days). | Rapid (4–12 hours). | Prodrome (1 decompens |
| w grade. | **High grade.** | Intermediat |
| **rking cough,** inspiratory stridor, arseness. | **Respiratory distress:** acute decompensation, toxic appearance, inspiratory stridor, muffled oice, drooling, tripoding. | Variable res slower onse pseudomem |
| one. | Seated, neck extended (tripod position). | None. |
| **ridor improves.** | None. | None. |
| **teeple sign"** on AP film | **"Thumbprint sign"** on lateral film | **Subglottic** |

young
igns on

- **Kernig sign** (reluctance of knee extension when the **Brudzinski sign** (hips are flexed in response to forced are nonspecific signs of meningeal irritation.
- Additional physical examination findings may inclu (papilledema, cranial nerve palsies) or a **petechial ras** Signs in neonates include lethargy, hyper- or hypoth **bulging fontanelle, and vomiting.**

Diagnosis

- Obtain a head CT to rule out ↑ ICP (risk of brainste **patient is at high risk** (ie, exhibits neurologic deficits on funduscopic exam)

| | |
|---|---|
| y unilateral; a mass may be<br>in the posterior pharyngeal<br>on visual inspection. | |
| **A streptococcus** (most<br>on), S aureus, Bacteroides (often<br>icrobial). | **Group A streptococcus** (most common), S aureus, S pneumoniae, anaerobes. |
| with the **neck extended**<br>up or fl xing the neck<br>ns symptoms). | None. |
| **eral neck x-ray film** the soft<br>plane should be ≤ 50% of the<br>of the corresponding vertebral<br>Contrast CT of the neck helps<br>ntiate abscess from cellulitis. | Usually **clinical.** |
| tion or incision and drainage<br>cess; antibiotics. | Incision and drainage ± tonsillectomy; antibiotics. |

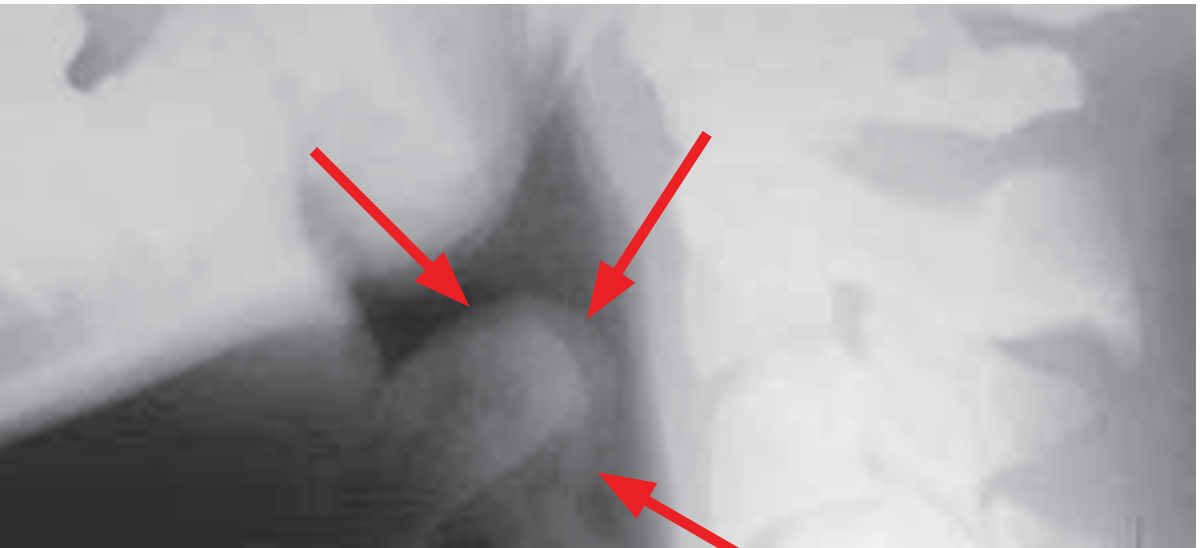


Table 2.13-16 summarizes the clinical presentation of co
lar infections.

### Diagnosis

- **Bacterial cultures** and Gram stain are the gold sta
  gonococcal or chlamydial conjunctivitis.
- **HSV PCR is the diagnostic standard for children**
  **ation or those with a vesicular eruption anywhere on**
- **The neonate's mother should undergo cervical Gra**
  **if STI is the suspected cause of conjunctivitis.**

### Treatment

- Empiric treatment can start before culture results are k
- For chlamydial conjunctivitis, give **topical erythrom**
  **oral erythromycin;** topical antibiotics alone are insu
  **infection is often present.**
- For gonococcal conjunctivitis, give an **IV/IM third**
  **losporin.** Coverage of gonococcus is crucial if the
  unknown, as **corneal ulceration** (and resultant scarrin
  **24–48 hours!**
- For HSV conjunctivitis, treat with a 14- to 21-day cou
  **clovir,** along with a topical agent (such as **vidarabine**).

## PERTUSSIS (WHOOPING COUGH)

A highly infectious form of bronchitis caused by the gr
detella pertussis. The DTaP vaccine (given in five doses
is protective, but immunity wanes by adolescence. Ado

**TABLE 2.13-16. Ocular Infections in the Neonatal Period**

**ussive emesis, and apnea.**

evated WBC count with lymphocytosis (often ≥ 70%).
**culture is the gold standard.**

nts < 6 months of age.
**cin** × 10 days to patients. Exposed newborns are at high of their immunization status because they may not be d by maternal transplacental immunoglobulins.
including daycare contacts) should receive prophylactic romycin × 5 days).

hes the clinical presentation of common viral exanthems.

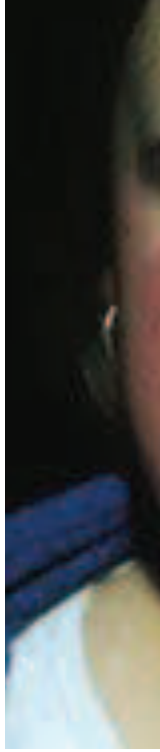


**FIGURE**
Note the
ance of
parvovir
from Wolff
Synopsis of
McGraw-H

**ral Exanthems**

| Cause | Characteristics | |
|---|---|---|
| Parvovirus B19 | **Prodrome:** None; fever is often absent or low grade.<br>**Rash: "Slapped-cheek,"** pruritic, maculopapular, erythematous rash (see Figure 2.13-17). Starts on the arms and spreads to the trunk and legs. **Worsens with fever and sun exposure.** | **Arthropathy** in<br>Congenital infe<br>hydrops and<br>**Aplastic crisis**<br>with ↑ RBC tu<br>hereditary sp<br>↓ RBC produc<br>anemia). |

| | | |
|---|---|---|
| | **In contrast to measles, children with rubella often have only a low-grade fever and do not appear as ill.** Polyarthritis may be seen in adolescents. | |
| nd -7 | **Prodrome:** Acute onset of high fever (> 40°C [> 104°F]); no other symptoms for 3–4 days.<br>**Rash: A maculopapular rash appears as fever breaks** (begins on the trunk and quickly spreads to the face and extremities) and often lasts < 24 hours. | **Febrile seizures** may res<br>onset. |
| | **Prodrome:** Mild fever, anorexia, and malaise precede the rash by 24 hours.<br>**Rash:** Generalized, pruritic, "teardrop" vesicles on red base; lesions are often at **dif erent stages of healing.** Usually appears on the face and spreads to the rest of the body, sparing the palms and soles.<br>Infectious from 24 hours before eruption until lesions crust over. | Progressive varicella with<br>pneumonia, and hepa<br>immunocompromised<br>Skin lesions may develop<br>Reye syndrome (associat |
| | **Prodrome:** Reactivation of varicella infection; starts as pain along an af ected sensory nerve.<br>**Rash:** Pruritic "teardrop" vesicular rash in a **dermatomal** distribution. Uncommon unless the patient is **immunocompromised.** | Encephalopathy, aseptic<br>pneumonitis, TTP, Guillai<br>cellulitis, arthritis. |
| ie A | **Prodrome:** Fever, anorexia, oral pain.<br>**Rash:** Oral ulcers; maculopapular vesicular rash | None (self-limited). |

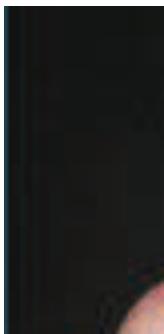
| | | | |
|---|---|---|---|
| | | | deaf |
| | Sexual contact, organ transplants. | Usually asymptomatic; mononucleosis-like illness. | Hear rash, peri |
| | Sexual contact, needlestick. | Variable presentation depending on CD4+ count. | Recu diarr |
| 2 | Skin or mucous membrane contact. | Usually asymptomatic; herpetic (vesicular) lesions. | Ence lesio |
| | Sexual contact. | Chancre (1°) and disseminated rash (2°) are the two stages likely to result in fetal infection. | Ofte fetal facia teeth sabe |

ssion from Le Tet al. First Aid for the USMLE Step 1 2015. New York, NY: McGraw-Hill; 2015.

## logy

em that helps evaluate the need for neonatal resuscitation
ch of five parameters is assigned a score of 0–2 at 1 and 5

Typically reflect good cardiopulmonary adaptation.
ndicate the possible need for resuscitation. Infants should



| | | |
|---|---|---|
| Appearance (skin color) | Completely pink. | Pink body with blue extremities. |
| Respirations | Vigorous cry. | Slow, irregular respirations. |

- **Conjugated (direct) hyperbilirubinemia:** Always path
- **Unconjugated (indirect) hyperbilirubinemia:** May pathologic. See Table 2.13-21 for differentiating charac
- **Kernicterus:** A complication of unconjugated hype results from irreversible bilirubin deposition in the and cerebellum. It typically occurs at levels of > 25–30 fatal. Risk factors include prematurity, asphyxia, and se

### History/Pe

See Table 2.13-22.

- The history should focus on diet (breast milk or for drug exposure, and family history (hemoglobinopathie cies, RBC defects).
- Physical examination may reveal signs of hepatic (abdominal distention, delayed passage of meconium, dark urine), infection, or birth trauma (cephalohemat lor, petechiae).
- **Kernicterus** presents with lethargy, poor feeding, hypertonicity, and seizures; jaundice may follow a ce sion as bilirubin concentrations ↑.

### Diagnosis

- For indirect hyperbilirubinemia, CBC with peripheral

suggestive; confi m with bronchoscopy.

**Treatment:** Surgical repair.

---

GI tract segments protrude through the diaphragm into the thorax; 90% are posterior left (Bochdalek).

**Presentation:** Respiratory distress (from pulmonary hypoplasia and pulmonary hypertension); sunken abdomen; **bowel sounds over the left hemithorax.**

**Diagnosis:** Ultrasound in utero; confi med by postnatal CXR.

**Treatment:** High-frequency ventilation or extracorporeal membrane oxygenation to manage pulmonary hypertension; surgical repair.

---

Herniation of the intestine only through the abdominal wall **next to the umbilicus (usually on the right) with no sac (the GI tract is exposed).**

**Presentation:** Polyhydramnios in utero; often premature; associated with GI stenoses or atresia. Presents with erythematous, matted bowel.

**Treatment:** Wrap exposed bowel with saline-soaked gauze, and secure with plastic immediately after birth. Surgical correction is needed in most cases.

---

Herniation of abdominal viscera through the abdominal wall **at the umbilicus into a sac** covered by peritoneum and amniotic membrane (see Figure 2.13-19).

**Presentation/diagnosis:** Polyhydramnios in utero; often premature; associated with other GI and cardiac defects. Seen in **Beckwith-Wiedemann syndrome and trisomies.**

**Treatment:** C-section can prevent sac rupture; if the sac is intact, postpone surgical correction until the patient is fully resuscitated. **Keep the sac covered/stable** with petroleum and gauze. Intermittent NG suction to prevent abdominal distention.



**FIGURE**
**locele in**
abdomin
nal wall
covered
membra
Brunicardi
ed. New Yo



| | |
|---|---|
| Resolves by 1 week in term infants and 2 weeks in preterm infants. | Persists beyond 1 wee 2 weeks in preterm inf |

phototherapy earlier (10–15 mg/dL) for preterm infan **not indicated for conjugated hyperbilirubinemia** a bronzing.

## RESPIRATORY DISTRESS SYNDROME

The most common cause of respiratory failure in **pret** > 70% of infants born at 28–30 weeks' gestation); former membrane disease. **Surfactant deficiency** leads to poo alveolar collapse, and atelectasis. Risk factors include r gender, and the second born of twins.

### History/Pe

Presents in the **first 48–72 hours of life** with a respiratory gressive hypoxemia, cyanosis, nasal flaring, intercostal ret tory grunting.

**s of Neonatal Jaundice**

| example(s) | |
|---|---|
| Hemolysis (ABO or Rh incompatibility). | ↑ u |
| Erythrocyte enzyme deficiency (G6PD deficiency). | |
| Erythrocyte structural defects (sickle cell anemia, hereditary spherocytosis). | |
| Ineffective erythropoiesis (thalassemias). | |

f > 30 weeks, monitor fetal lung maturity via a lecithin-
n (L/S) ratio and the presence of phosphatidylglycerol in

An L/S r
materna

**onchopulmonary dysplasia, retinopathy of prematurity,**
ositive pressure ventilation, intraventricular hemorrhage,
lications of treatment.

## Neurologic Disease

**ditary, nonprogressive disorders of movement and pos-**
mon movement disorder in children. Often results from
**insult,** but in most cases the cause is unknown. Risk fac-
rth weight, intrauterine exposure to maternal infection,
tal asphyxia, trauma, brain malformation, and neonatal
e. Categories include the following:

**tic):** Spastic paresis of any or all limbs. Accounts for 75%
tual disabilities are present in up to 90% of cases.
**(dyskinetic):** A result of damage to extrapyramidal tracts.
xic (difficulty coordinating purposeful movements), cho-
dystonic (uncontrollable jerking, writing, or posturing).
ments worsen with stress and disappear during sleep.

**R Findings in Neonatal Lung Pathology**

| Process | Key Findings |
|---|---|



B C

**lung pathologies. (A)** Chest radiograph of an intubated infant illustrating classic fin
r or ground glass appearance of the lung parenchyma, the poor inflation, the lack of
Two-day-old child with transient tachypnea of the newborn, exhibiting perihilar stre
othorax and coarse, irregular infiltrates in a neonate with meconium aspiration. (Part A
Medicine: A Comprehensive Study Guide, 7th ed. New York, NY: McGraw-Hill; 2010. Part B reproduced with permis
iabetic mothers, Indian J Radiol Imaging 2010 Aug;20(3):174-181. Part C reproduced with permission from Khan
st radiographic appearance, instrumentation and complications from instrumentation, Ann Thorac Med 2009 A

ayed

### History/Pe

- May be associated with seizure disorders, behavioral c
  vision impairment, learning disabilities, and speech de
- Affected limbs may show hyperreflexia, pathologic ref
  ↑ tone/contractures, weakness, and/or underdevelopn
  preference before 1 year of age is a red flag.
- Toe walking and scissor gait are common. Hip dislo
  may be seen.

### Diagnosis

Diagnosed by clinical impression, although imaging ca
mine the underlying cause of CP in some cases. For ins
phy may be useful in infants to identify intracranial hemo
malformations, and MRI is diagnostic in older children.
in patients with suspected seizures.

### Treatment

- There is **no cure for cerebral palsy.** Special educatio
  braces, and surgical release of contractures may help.
- Treat spasticity with diazepam, dantrolene, or baclofe
  and posterior rhizotomy may alleviate severe contractu

fection (eg, altered consciousness, meningismus, a tense/
fontanelle) after ruling out ↑ ICP.
ecessary for first-time simple febrile seizures, and no lab
ed if presentation is consistent with febrile seizures in chil-
ns of age. Infants < 6 months of age need a sepsis workup
blood, urine, and CSF culture).
sentations, obtain electrolytes, serum glucose, blood cul-
BC with differential.

**therapy** (acetaminophen; **avoid ASA in light of the risk**
**ne**) and treat any underlying illness. Note that antipyretic
↓ the recurrence of febrile seizures.
zures, perform a thorough neurologic evaluation, includ-
an MRI. Chronic anticonvulsant therapy (eg, diazepam
) may be necessary.

rrence is < 30% and is highest within 1 year of the initial
ple febrile seizures, there is no ↑ risk of developmental
epilepsy.
he development of epilepsy include complex febrile sei-
k), ⊕ family history of epilepsy, an abnormal neurologic
opmental delay.

## Oncology

alignancy of lymphocytic or myeloblastic origin. The most

Perform
suspect
seizure.

Simple f
brain da
do **not**

- **CXR to rule out a mediastinal mass.**

### Treatment

- Chemotherapy based, including induction, consolid
  nance phases.
- **Tumor lysis syndrome** is common during the initiat
  cancers with high cell turnover (such as leukemias
  is caused by the lysis of many neoplastic cells in a sh
  resulting in the release of cell contents into the bloo
  acterized by hyperkalemia, hyperphosphatemia, hyper
  calcemia (as calcium is bound by phosphate released
  tic cells). It can result in renal failure, arrhythmias, an
  fluids, diuretics, and allopurinol (which reduces the r
  nephropathy). **Corticosteroids may** precipitate tumor l

at the
men.

## NEUROBLASTOMA

An embryonal tumor of neural crest origin. More than ha
**years of age,** and 70% have distant metastases at presentat
**neurofibromatosis, Hirschsprung disease,** and the N-myc

### History/Pe

- Lesion sites are most commonly abdominal, thoraci
  descending order).
- Symptoms may vary with location and may include a
  **inal mass (may cross the midline), Horner syndrom**
  cord compression (from a paraspinal tumor).
- Patients may have anemia, FTT, and fever.
- More than 50% of patients will have metastases at diag
  bone marrow suppression, proptosis, hepatomegaly, su
  (Figure 2.13-22), and **opsoclonus/myoclonus.**



**Neuroblastoma presenting with multiple subcutaneous nodules.**
the Armed Forces Institute of Pathology, *Atlas of Tumor Pathology.*)

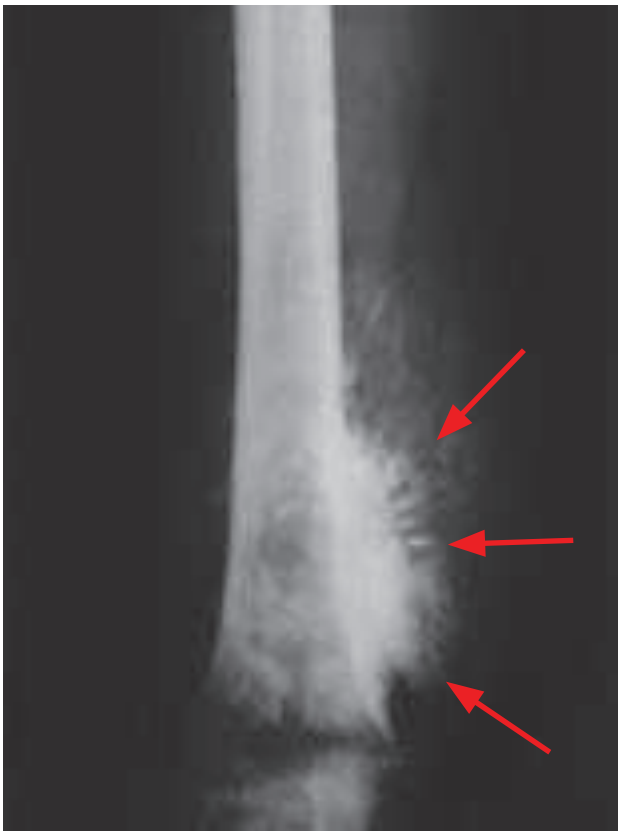
mbryonal origin that is most commonly seen in **children** sociated with **Beckwith-Wiedemann syndrome** (hemihy-ssia, visceromegaly), **neurofibromatosis, and WAGR syn-**or, **A**niridia, **G**enitourinary abnormalities, mental **R**etar-"intellectual disabilities").

**symptomatic, nontender,** smooth abdominal mass that **cross the midline.**
, fever, hypertension, and microscopic or gross hematuria

edle aspiration is required for definitive diagnosis.
sonography.
chest and abdomen are used to detect metastases.

nephrectomy with postsurgical chemotherapy and radia-tage and histology.

| | | |
|---|---|---|
| History/PE | Local pain and swelling.<br>**Systemic symptoms** (fever, anorexia, fatigue) are common. | Local pain<br>Systemic s |
| Location | **Midshaft** of long bones (femur, pelvis, fibula, humerus) | **Metaphys**<br>femur, p<br>humerus<br>Metastases |
| Diagnosis | Leukocytosis, ↑ ESR.<br>Lytic bone lesion with **"onion skin"** periosteal reaction on plain x-ray film | ↑ **alkaline**<br>**"Sunburst**<br>Chest CT t<br>metasta |
| Treatment | Local excision, chemotherapy, and radiation. | Local excis |





| | |
|---|---|
| Incomplete fracture involving the cortex of only 1 side (tension/trauma side) of the bone. | Reduction with ca |
| **Radial head subluxation.** Secondary to being pulled or lifted by the hand. Pain, pronation, and refusal to bend the elbow. | **Manual reductio** elbow at 90 degre |
| **Buckling** of the compression side of the cortex 2° to trauma. Usually occurs in the distal radius or ulna as a result of a fall. | Cast immobilizati |
| **The most common pediatric elbow fracture.** Tends to occur at 5–8 years of age. Proximity to the brachial artery ↑ the **risk of Volkmann contracture** (results from compartment syndrome of the forearm).<br>Beware of **brachial artery entrapment** (check radial pulse).<br>See Figure 2.13-24. | Cast immobilizati percutaneous pin |
| **Overuse** apophysitis of the tibial tubercle. Causes localized pain, especially with quadriceps contraction, in active young boys. | ↓ activity for 2–3 Neoprene brace f |
| Fractures of the growth plate in children. Classified y fracture pattern:<br>**I:** Physis (growth plate).<br>**II:** Metaphysis and physis.<br>**III:** Epiphysis and physis.<br>**IV:** Epiphysis, metaphysis, and physis.<br>**V:** Crush injury of the physis. | Closed vs open re alignment, follow |

LAR DYSTROPHY



Physical therapy is necessary to maintain ambulation and
tures. Liberal use of tendon release surgery may prolong a

### Complications

Mortality is due to high-output cardiac failure (stemming
sis).

## DEVELOPMENTAL DYSPLASIA OF THE HIP

Also called congenital hip dislocation; can result in sub
femoral heads, leading to early degenerative joint disease
from poor hip development due to lax musculature an
**packing** in flexed and adducted position (eg, breech pres
excessive stretching of posterior hip capsule and contractu

### History/Pe

- Most commonly found in **firstborn girls** born in th
  ↑ risk with family history.
- **Barlow maneuver:** Posterior pressure is placed on
  abducted thigh, and hip is then adducted, leading to a
  the femoral head dislocates posteriorly.
- **Ortolani maneuver:** Thighs are gently abducted fro
  anterior pressure on the greater trochanter. **Soft click**
  of femoral head into acetabulum.
- **Allis (Galeazzi) sign:** The knees are at unequal height
  knees are flexed (the dislocated side is lower).
- **Asymmetric inguinal skin folds** that extend beyond t
  abduction of the affected hip are also seen.

**TABLE 2.13-26. DMD vs Becker Muscular Dystrophy**

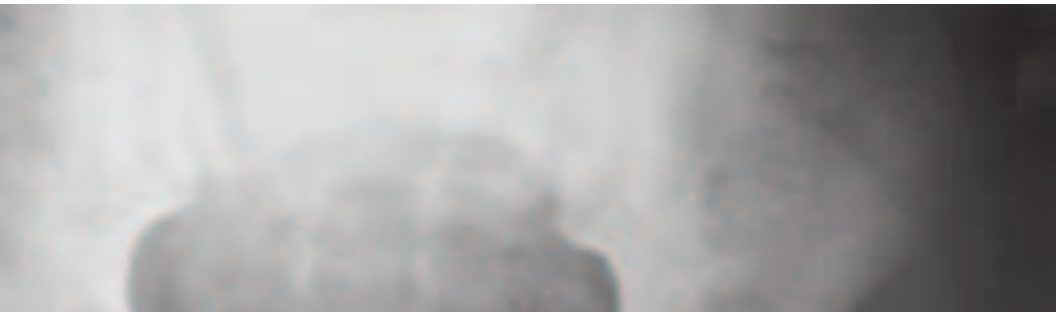
pica cast.
Open reduction followed by spica cast.

s and AVN of the femoral head.
nt, a significant defect is likely in patients < 2 years of age.

## HES DISEASE

d osteonecrosis of the femoral head (see Figure 2.13-25). oys 4–10 years of age. Usually a self-limited disease, with 18 months.

tomatic at first, but patients can develop a **painless limp,** thigh muscle atrophy.
t, it can be in the groin or anterior thigh, or it may be nee.





- If extensive or ↓ ROM, consider bracing, hip abductio or an osteotomy.
- The prognosis is good if the patient is < 6 years of age ↓ femoral head involvement, and a stable joint.

## SLIPPED CAPITAL FEMORAL EPIPHYSIS

Displacement of the femoral epiphysis from the femora growth plate. The name is misleading, because the epiph the acetabulum while the metaphysis moves anteriorly sents in **obese** children ages 10–16. Associated with hypot endocrinopathies.

### History/Pe

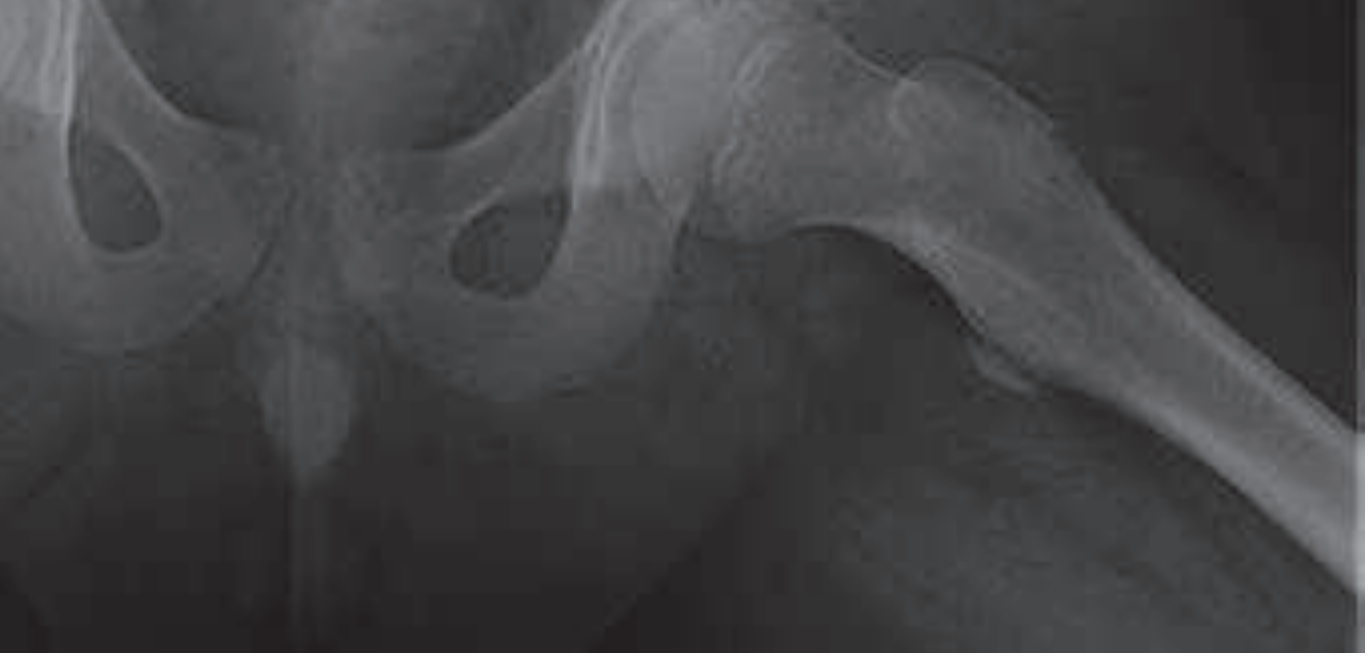
- Insidious onset of dull hip pain, or referred knee pain,
- Restricted ROM and **inability to bear weight** (differen stable SCFE).
- Bilateral in 40–50% of cases.
- Limited internal rotation and abduction of the hip. I passive external rotation.

### Diagnosis

- Radiographs of **both** hips in **AP and frog-leg lateral vi and inferior displacement** of the femoral head (see Fi
- In patients under the 10th percentile of height, rule with TSH.

### Treatment

- The disease is progressive, so treatment should begin p
- Immediate surgical screw fixation to reduce risk of AV
- **No weight bearing** should be allowed until the defec lized.



**lipped capital femoral epiphysis.** Frog-leg AP radiograph demon-
or displacement of the right femoral epiphysis (red arrow) relative to
nparison, the left side (blue arrow) is normal. (Reproduced with permission

e is usually identified during school physical screening.
p rotation deformities are accentuated by the Adams for-
st.

spine (posterior, anterior, and full-length views).

n for < 20 degrees of curvature.
or 20–49 degrees of curvature in patients with remaining
**re may progress even with bracing.**
on for > 50 degrees of curvature.

## HEARING AND VISION SCREENING

- Objective hearing screening (otoacoustic emissions an
  stem response) for newborns prior to discharge is comm
- Objective hearing screening is indicated for childre
  meningitis, ToRCHeS infections, measles and mump
  tis media.
- The red reflex should be checked at birth. Leukocoria
  reflex and can indicate the presence of retinoblastor
  incidental finding in a baby's first photos.
- **Strabismus** (ocular misalignment) is normal until
  beyond 3 months, children should be evaluated by
  mologist and may require corrective lenses, occlusion
  prevent **amblyopia** (suppression of retinal images in
  leading to permanent vision loss).

## CHILDHOOD VACCINATIONS

The Epidemiology chapter summarizes CDC-recommen
the pediatric population. Contraindications and precaut
tion are as follows:

- **Contraindications:**
  - Severe allergy to a vaccine component or a pri
    **Patients who have life-threatening allergies to eg
    MMR.**
  - Encephalopathy within 7 days of prior pertussis vac
  - Avoid live vaccines (oral polio vaccine, varicella,
    compromised and pregnant patients (exception:
    receive MMR and varicella).
- **Precautions:**
  - Current moderate to severe illness (with or without
  - Prior reactions to pertussis vaccine (fever > 40.5
    shocklike state, persistent crying for > 3 hours with

test as an initial screen at age 1 or 2; then obtain serum

eral blood smear show **microcytic, hypochromic anemia stippling (Figure 2.13-27). Sideroblastic anemia may**

**asymptomatic:** Retest at 1–3 months; remove sources of

helation therapy (inpatient **EDTA** or outpatient oral suc-

elation therapy (inpatient **EDTA** + **BAL** [IM dimercap-

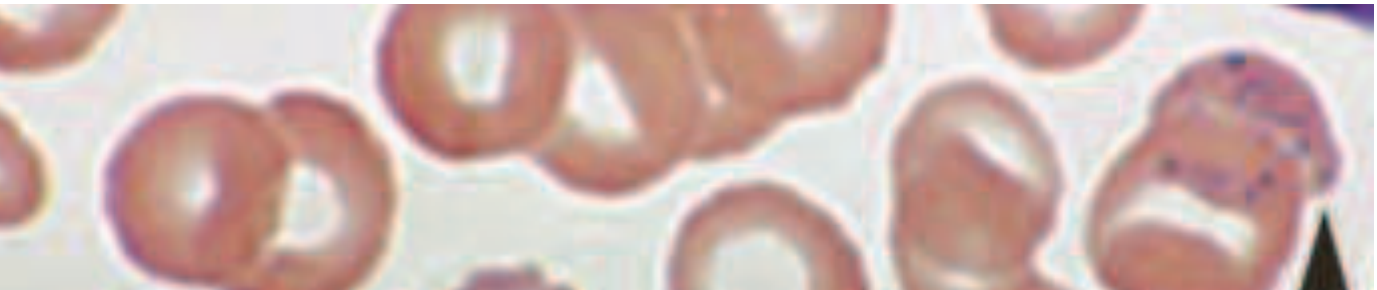
## Urology

### REFLUX

on of urine from the bladder to the ureters and kidneys. erior urethral valves, urethral or meatal stenosis, or a neu- assified as follows:

**ades I–II):** No ureteral or renal pelvic dilation. Often eously.

Posterio
most co
obstruct
male inf
bladder





VCUG should also be performed if there is recurrence of

### Treatment

- Treat infections aggressively. Treat mild reflux with antibiotics (amoxicillin if < 2 months of age; other nitrofurantoin) until reflux resolves.
- Surgery (ureteral reimplantation) is generally reserve persistent high-grade (III to V) reflux. Inadequate tre progressive **renal scarring** and end-stage renal disease.

## CRYPTORCHIDISM

Failure of one or both of the testes to fully descend into **birth weight is a risk factor.**

### History/Pe

Bilateral cryptorchidism is associated with prematurity, ol ital malformation syndromes (Prader-Willi, Noonan synd ity. Associated with an ↑ **risk of testicular malignancy.**

### Diagnosis

The testes **cannot be manipulated into the scrotal sac** (vs retractile testes) and can be palpated anywhere along t in the abdomen.

### Treatment

- **Orchiopexy** for prepubertal boys; orchiectomy if disc to minimize the risk of testicular cancer.
- If discovered later, consider orchiectomy to prevent close observation.

cent Disorders 422
Ac TIVITY d ISo Rd ER 422
ER 422
ISo Rd ERS 423
NTAL d ISo Rd ER 423
423
424
424
425
o Rd ER 425
426
c IFIc ) 427
Disorder and Related Disorders 428
ISo Rd ER 428
d ER 429
elated Disorders 429
ISo Rd ER 429
430
ers 430
EURo c o GNITIVE d ISo Rd ER) 430
431
Mood Disorders
MAJo R d EPRESSIVE d ISo Rd ER
BIPo LAR d ISo Rd ER
Personality Disorders
Substance Use Disorders
ALc o Ho L USE d ISo Rd ER
Eating Disorders
ANo REXIA NERVo SA
BULIMIA NERVo SA
Miscellaneous Disorders
SEXUAL d ISo Rd ERS
SLEEP d ISo Rd ERS
So MATIc SYMPTo M ANd RELATEd d ISo
FAc TITIo US d ISo Rd ERS ANd MALINGERI
SEXUAL ANd PHYSIc AL ABUSE
SUIc Id ALITY

Diagnosis requires **five or more symptoms** from each cate
**6 or more months** in **at least two settings,** leading to si
academic impairment. Symptoms must be present in patie

- **Inattention:** Exhibits a **poor attention span** in schoo
  poor attention to detail or careless mistakes; has
  **instructions or finishing tasks;** is forgetful and easily d
- **Hyperactivity/impulsivity: Fidgets;** leaves seat in clas
  inappropriately; cannot play quietly; talks excessively
  **his or her turn; interrupts others.**

### Treatment

- Initial treatment may be nonpharmacologic (eg, beh
  Sugar and food additives are **not** considered etiologic f
- Pharmacologic treatment includes the following:
  - **Psychostimulants: Methylphenidate (Ritalin),**
    (Dexedrine), mixed salts of dextroamphetamine
    (Adderall). Adverse effects include insomnia, irrital
    exacerbation, and ↓ growth velocity (normalizes
    stopped).
  - **Nonstimulants** (eg, SSRIs, nortriptyline, bupropi
    (eg, **clonidine**), and atomoxetine (Strattera).

## AUTISM SPECTRUM DISORDER

Persistent impairment in socialization, communication,
common in males. Symptom severity and IQ vary widel
distinguish between the pervasive developmental disorde
on the level of support needed in the psychopathologi
communication and restricted/repetitive behaviors.

### History/Pe

- Characterized by abnormal or impaired social interacti

or age-appropriate **societal norms or rules** for **1 year or** may be aggressive (eg, rape, robbery, animal cruelty) or eg, stealing, lying, deliberately annoying people). **May ocial personality disorder in adulthood.**

ly therapy.

## VELOPMENTAL DISORDER

le gender, chromosomal abnormalities, congenital infec- ncluding alcohol/illicit substances), and inborn errors of

paired intellectual functioning **(IQ < 70)** with **deficits in ning** (eg, hygiene, social skills); onset is before age 18. ty are **mild (IQ 50–70; 85% of cases),** moderate **(IQ Q 20–34),** and profound **(IQ < 20).**

onsists of educating the general public about possible ctual disability and providing optimal prenatal screening

ures include family counseling and support; speech and y; occupational/physical therapy; behavioral intervention; tance; and social skills training.

## ME

nen and boys; shows a genetic predisposition. **Associated**

Conduc
Antisoc
in Adult

Fetal alc
commo
disabilit

**women (ages 25–35).** Schizophrenia in first-degree r
Up to 50% of patients attempt suicide, and **10% of tho**
**fully commit suicide.**

- **Etiology:** Etiologic theories focus on neurotransmitter
as dopamine dysregulation (frontal hypoactivity and l
and brain abnormalities on CT and MRI (enlarged ve
cal volume).

## History/Pe

- Two or more of the following are present continuo
**months** with **social or occupational dysfunction:**
  - **Positive symptoms:** Hallucinations (most often a
disorganized speech, bizarre behavior, and thought
  - **Negative symptoms:** Flat affect, ↓ emotional re
speech, lack of purposeful actions, and anhedonia.
- See Table 2.14-1 for the differential diagnosis of psycho

**TABLE 2.14-1. Differential Diagnosis of Psychosis**

| Disorder | Duration/Characteristi |
|---|---|
| Psychotic disorders | **Brief psychotic disorder:** > 1 day and < 1 m<br>**Schizophreniform disorder:** > 1 month a<br>Note: Both have same presentation as schizo<br>preceded by stressors, have no prior episo<br>have negative symptoms, and have better<br>**Schizophrenia:** > 6 months.<br>**Schizoaffective disorder:** Schizophrenia + m<br>(major depressive disorder or bipolar affec |
| Personality disorders | **Schizotypal:** "Magical thinking." |

**cessive anxiety or worry about multiple activities or** significant impairment or distress. The male-to-female onset is usually in the early 20s.

**psychotic Medications**

| | Examples | Indications |
|---|---|---|
| gh potency. | Haloperidol, fluphenazin . | Psychotic disorders, acute agitation, acute mania, Tourette syndrome.<br>Thought to be more effective for positive symptoms of schizophrenia; primarily block D2 dopamine receptors.<br>For patients in whom compliance is a major issue, consider antipsychotics that come in depot forms (eg, haloperidol, fluphenazine) |
| w potency. | Thioridazine, chlorpromazine. | Same as high potency. |

| | | |
|---|---|---|
| igidity). | | agonist (amantadine). ↓ the dose discontinue (if tolerated). |
| /objective restlessness that is as being distressing. | Weeks | ↓ neuroleptic and try β-blockers ( Benzodiazepines or anticholinerg |
| c, involuntary, painless oral-facial s. Probably from dopamine receptor n from chronic dopamine blockade. ersible (50%). | Months | Discontinue or ↓ the dose of neur treatment with more appropriate changing neuroleptic (eg, to cloz **Giving anticholinergics or decr may initially worsen tardive dys** |
| cle rigidity, autonomic instability, Kand WBC, clouded consciousness. | Anytime | **Stop medication;** provide suppo administer dantrolene or bromoc |

His To r y/Pe

Presents with anxiety on most days **(6 or more months) more somatic symptoms** (restlessness, fatigue, difficulty c bility, muscle tension, disturbed sleep).

Tr ea Tmen T

- **Short-term therapy:**
  - Benzodiazepines may be used for immediate sympt
  - Taper benzodiazepines as soon as long-term treat (eg, with SSRIs) in view of the high risk of toleranc
  - Do not stop benzodiazepines "cold turkey," as pa potentially lethal withdrawal symptoms similar to th drawal.
- **Long-term therapy:**

| | | |
|---|---|---|
| | Phobic disorders (best when given prior to exposure). | Bradycardia, hypotension. |
| | Anxiety, insomnia, alcohol withdrawal, muscle spasm, night terrors, sleepwalking. | ↓ sleep duration; risk of abuse, tolerance, and dependence; disinhibition in young or old patients; confusion. |

e defined as discrete **periods of intense fear or discom-**
least four of the following symptoms develop abruptly and
minutes: tachypnea, chest pain, **palpitations, diaphoresis,**
g, dizziness, **fear of dying** or "going crazy," depersonali-
shes.
cral paresthesias, when present, are fairly specific to panic
oduce hyperventilation and low $O_2$ saturation.
with **1 or more months** of concern about having addi-
significant behavior change as a result of the attacks—eg,
ns that may precipitate attacks.
**ther a patient has panic disorder with or without agora-**
goraphobia can also be addressed in the treatment plan.

Walks lik
… it's a
panic di
- **Me**
  MI, a
  phe
- **Psy**
  Sub
  PTSI

**apy:** Benzodiazepines (eg, clonazepam) may be used
elief, but long-term use should be avoided in light of the
iction and tolerance (see Table 2.14-4). Taper benzodiaz-
long-term treatment is initiated (eg, SSRIs).
**py:**

exposure to the feared object or situation along with re
Other options include supportive, family, and insight-
apy.

- **Social phobias: CBT,** SSRIs, low-dose benzodiazepin
performance anxiety) may be used (see Table 2.14-4).

# Obsessive-Compulsive Disorder and Related Disorders

## OBSESSIVE-COMPULSIVE DISORDER

Characterized by obsessions and/or compulsions that lea
tress and dysfunction in social or personal areas. Typica
adolescence or early adulthood; prevalence is equal in
patients. Often chronic and difficult to treat.

### History/Pe

- **Obsessions: Persistent, unwanted, and intrusive
impulses, or images** that lead to marked anxiety or
contamination, fear of harm to oneself or to loved ones
- **Compulsions: Repeated mental acts or behaviors** t
ety from obsessions (eg, handwashing, elaborate ritua
counting, excessive checking).
- Patients **recognize these behaviors as excessive and
of their own minds** (vs obsessive-compulsive pers
OCPD; see Table 2.14-5).
- Patients wish they could get rid of the obsessions and/o

**TABLE 2.14-5. OCD vs OCPD**

sons) in response to concerns of appearance.

## nd Stressor-Related Disorders

### TRESS DISORDER

t stress or impairment in day-to-day social/work interac-
direct exposure to an extreme, **life-threatening traumatic**
ombat, injury, rape, accident, violent crime), witnessing a
irect exposure through learning of a life-threatening event
mily member or friend, or reexposure to trauma-related
pation.

Top cau
are (1) s
Top cau
are (1) c
assault.

y the following four symptom clusters:
**eexperiencing of the event** through nightmares, flash-
ve memories.
stimuli associated with the trauma.
**erations in mood and cognitions: Numbed responsive-**
chment, anhedonia), guilt, self-blame.
**arousal and reactivity: ↑ arousal** (eg, hypervigilance, exag-
e), sleep disturbances, aggression/irritability, and poor
that leads to significant distress or impairment in func-

**persist for > 1 month;** the same symptoms present for 3
characterize acute stress disorder.

onset.

### Treatment

**No pharmacologic treatment!** Treat adjustment disor
counseling.

## Neurocognitive Disorders

Affect memory, orientation, judgment, and attention.

### DEMENTIA (AKA MAJOR NEUROCOGNITIVE DISORDER)

A decline in **cognitive** functioning with **global deficits.**
**ness is stable (vs delirium).** Prevalence is highest among
age. The course is persistent and progressive. The most
**Alzheimer disease** (65%) and **vascular dementia** (20%
outlined in the mnemonic **DEMENTIASS.**

### History/Pe

Diagnostic criteria include **memory impairment and one**
lowing:

- **The four A's of dementia** (the progression of cognit
lows this order): **A**mnesia (partial or total memory loss)
impairment), **A**praxia (inability to perform motor
(inability to recognize previously known objects/places
- **Impaired executive function (problems with planni**
**abstracting)** in the presence of a **clear sensorium.**
- Personality, mood, and behavior changes are common
aggression).

| | |
|---|---|
| uded. | Intact. |
| sent (often visual or tactile). | Occur in approximately 30% of patients in highly advanced disease. |
| versible. | Largely irreversible, but up to 15% of cases are due to treatable causes and are reversible. |
| at underlying causes.<br>w-dose antipsychotics;<br>environmental changes (ie, low-timulus environment, frequent orientation to day and time, hades up during daytime to eestablish circadian rhythm). | Cholinesterase inhibitors; low-dose antipsychotics (primarily for behavior disturbances).<br>Environmental changes. |

**nhibitors** are used to treat. Low-dose **antipsychotics** may hotic symptoms and sometimes for aggression, but with f cardiovascular events in elderly patients. **Avoid benzodi-** may exacerbate disinhibition and confusion.
r, and patient education and support are imperative.

**ce of consciousness** with **altered cognition** that develops of time (usually hours to days). Children, the elderly, and s (eg, **ICU psychosis**) are particularly susceptible. Major

Major c
**IWATC**
Infection

- **Treat underlying causes** (delirium is often reversible).
- Normalize fluids and electrolytes.
- **Optimize the sensory environment, and provide n hearing aids.**
- Use low-dose **antipsychotics** (eg, haloperidol) for agit symptoms.
- Conservative use of **physical restraints** may be necess to the patient or others.

# Mood Disorders

Also known as **affective disorders.**

## MAJOR DEPRESSIVE DISORDER

A mood disorder characterized by one or more major (MDEs). The male-to-female ratio is 1:2; lifetime prev 15% to 25%. **Onset is usually in the mid-20s;** in the ↑ with age. Chronic illness and stress ↑ risk. Approximat die by suicide.

### History/Pe

Diagnosis requires **depressed mood or anhedonia (loss and five or more signs/symptoms** from the **SIG E CAl 2-week period** (you should be sure to learn this mnemon questions). Table 2.14-7 outlines the differential diagnosis o be mistaken for depression. Selected depression subtypes inc

- **Psychotic features:** Typically **mood-congruent** delusio
- **Postpartum:** Occurs **within 1 month postpartum;** h and a high risk of recurrence. Psychotic symptoms are 2.14-8).

| | |
|---|---|
| | A constellation of symptoms that resemble an MDE but do not meet the criteria for MDE. Occurs within 3 months of an identifiable st essor. |
| | Occurs after the loss of a loved one. Involves no severe impairment/suicidality. Usually lasts < 6 months; should resolve within 1 year. May lead to MDD that requires treatment. Illusions/hallucinations of the deceased can be normal as long as the person recognizes them as such. |
| | Milder, chronic depression with depressed mood present most of the time for at least 2 years; often resistant to treatment. |

**Psychotherapy combined with antidepressants is more ther treatment alone.**

**ve therapy (ECT):**

effective, and often lifesaving therapy that is reserved for pression or psychotic depression, if rapid improvement in ded (severe suicidality), or intractable mania and psycho- during pregnancy.

ires two to three times per week treatment for a total of nts.

cts include postictal confusion, arrhythmias, headache, **ade amnesia.**

contraindications. Relative contraindications include oke, intracranial mass, and high anesthetic risk.

Disconti
before s
the pati

**erential Diagnosis of Postpartum Disorders**

| tiMe oF On Set | SyMpto MS |
|---|---|

| | | |
|---|---|---|
| zapine, | Depression, anxiety. | **Bupropion:** ↓ seizure threshold; minimal sexua<br>Contraindicated in patients with eating disor<br>patients.<br>**Mirtazapine:** Weight gain, sedation.<br>**Trazodone:** Highly sedating; priapism. |
| xetine. | Depression, anxiety, chronic pain. | **Venlafaxine:** Diastolic hypertension. |
| pramine. | Depression, anxiety disorder, chronic pain, migraine headaches, enuresis (imipramine). | Lethal with overdose resulting from cardiac con<br>(eg, prolonged conduction through the AVn<br>in the ICU for 3–4 days following an overdose<br>Anticholinergic effects (dry mouth, constipation<br>sedation). |
| h form is | Depression, especially atypical. | Hypertensive crisis if taken with high-tyramine<br>wine).<br>Sexual side effects, orthostatic hypotension, we |

= BPD.

- **Phototherapy:** Effective for patients whose depression ha
- **Transcranial magnetic stimulation (TMS):** Now app
ment of major depression. TMS is about as effective
some patients but is not as effective as ECT.

## BIPOLAR DISORDER

Prevalence is approximately 1% for type I and an additi
male and female patients are affected equally. A family h

mania.
ot due to a substance or medical condition and lead to sig-
ent socially, occupationally, or familially.
milar but does not involve marked functional impairment
ptoms and does not require hospitalization.

Sexual in
activit
Talkative

Mania is considered a psychiatric emergency because of
gment and great risk of harm to self and others.
**y:** Antipsychotics, lithium, valproate.
**e therapy:** Mood stabilizers (see Table 2.14-10).
zepines for refractory agitation.
**ion:** Mood stabilizers with or without antidepressants.
**ilizers first** (see Table 2.14-10) to avoid inducing mania.
d to treat refractory cases.

**ood Stabilizers**

| | Indications | Sid |
|---|---|---|
| | rst-line mood stabilizer. Used for acute mania (in mbination with antipsychotics), for prophylaxis in BPD, d for augmentation in depression treatment. Also ecreases suicide risk. | Thirst, polyuria, diabetes i gain, hypothyroidism, n teratogenicity (if used in Ebstein anomaly), acne, Narrow therapeutic windo monitored). Lithium toxicity: blood lev ataxia, dysarthria, deliriu Avoid lithium in patient |
| | cond-line mood stabilizer; anticonvulsant; trigeminal | Nausea, skin rash, leukope |

affect most aspects of one's life (see the mnemonic **ME**
by early adulthood. Specific disorders are outlined in Tabl

**Symptoms of Personality Disorders**

| Characteristics | Clinical Presentat |
|---|---|
| **Cluster A: "Weird"** | |
| icious; interpret others' motives as | 59-year-old man who lives alone consta neighbor's children are spying on him a into his home. He has installed security property to obtain proof. He feels he ca do a good job because they will probab neighbors. |
| ed "loners." Restricted emotional | 66-year-old man who moves to Thailand retirement, has no desire to remain in c and is very distant in his interactions. He accommodations without unnecessary crave interaction with the locals. |
| erceptions, and appearance. **Magical** as of reference.<br>ents may have eccentric/disorganized behavior; however, they lack delusions and s that would classify them as schizophrenic. | 35-year-old man with very strange ideas importance of crystals and their effect o meticulously mines and collects crystals one day prevent him from acquiring ca |
| **Cluster B: "Wild"** | |
| , relationships, and self-image; feelings of | 28-year-old woman presents to clinic af |

| | |
|---|---|
| | all wasting his time. |
| rights of others, social norms, and laws. Impulsive; morse. **Must have a prior diagnosis of conduct er.** | 22-year-old man who was in ju teenager and presents to your a brutal assault. He says that he a shrink and that the victim off assault. |
| **Cluster C: "Worried and Wimpy"** | |
| upied with perfectionism, order, and control at the ense of efficie y. Infl xible morals and values. Remember, in contrast to OCD, these patients don't their behavior is problematic. | 35-year-old woman presents to her boss, who feels she is too fo team projects and doesn't allo of unwanted errors. She doesn style of work, as she believes h pay adequate attention to deta |
| y inhibited; rejection sensitive. **Fear of being d or ridiculed, yet desires to have friends and interactions.** | 33-year-old man stays at home party, as he fears having to ma though is more afraid that he w others. |
| ssive, clingy; have a need to be taken care of. Have y making decisions. Feel helpless. | 30-year-old woman presents to that her parents just kicked her struggling to survive on her ow even make choices at the groc care for her and now these dec has been sitting outside of the let her live there again. |

- Failed attempts to cut down use or abstain from the
- Increased amount of time spent acquiring, using, effects.
- Craving.

- Social impairment:
  - **Failure to fulfill responsibilities** at work, school, o
  - Continued substance use despite recurrent soci problems 2° to the effects of such use (eg, frequ spouse over the substance use).
  - **Isolation** from life activities.
- Risky use:
  - Use of substances in **physically hazardous** situatio intoxicated).
  - Continued substance abuse despite recurrent physi problems 2° to the effect of the substance use.
- Pharmacologic:
  - **Tolerance** and use of progressively larger amounts desired effect.
  - **Withdrawal** symptoms when not taking the substan

Note: Tolerance and withdrawal are not needed to mal

### Diagnosis/Treatment

- Substance use is often denied or underreported, so information from family and friends.
- Severity is determined by number of symptoms present moderate, four to five; severe, more than six.
- Check urine and blood toxicology screens, LFTs, and
- The management of intoxication for selected drugs is 2.14-12.

## ALCOHOL USE DISORDER

Occurs more often in **men** (4:1) and in those 21–34 years

| | |
|---|---|
| aloxone and naltrexone block opioid receptors and reverse effects, but beware of the antagonist clearing before the opioids, particularly with long-acting opioids such as methadone. | diarrhea, yawning.<br>Opioid withdrawal is not and does not cause sei |
| sychomotor agitation, impaired judgment, hypertension, **pupillary dilation,** tachycardia, fever, diaphoresis, anxiety, angina, euphoria, prolonged wakefulness/ attention, arrhythmias, delusions, seizures, hallucinations.<br>aloperidol can be given for severe agitation and symptom-targeted medications (eg, antiemetics, NSAIDs). | Postuse "crash" with anxie cramps, hunger, fatigue, disturbance, nightmares. |
| sychomotor agitation, euphoria, impaired judgment, tachycardia, **pupillary dilation,** hypertension, paranoia, hallucinations, "cocaine bugs" (the feeling of bugs crawling under one's skin), sudden death. ECG changes from ischemia are often seen ("cocaine chest pain").<br>eat with haloperidol for severe agitation along with symptom-specific medic tions (eg, to control hypertension). | Postuse "crash" with hype malaise, severe craving, a nightmares. |
| **ssaultiveness,** belligerence, psychosis, violence, impulsiveness, psychomotor agitation, fever, tachycardia, **vertical/horizontal nystagmus,** hypertension, impaired judgment, ataxia, seizures, delirium.<br>ive benzodiazepines or haloperidol for severe symptoms; otherwise reassure. Acidific tion of urine or gastric lavage can help eliminate the drug. | Recurrence of intoxicatio in the GI tract; sudden on |
| larked anxiety or depression, delusions, visual | None. |

| | |
|---|---|
| ss, insomnia, diuresis, muscle twitching, as, tachycardia, flushed fa e, psychomotor | Headache, lethargy, depression, w craving. |
| ss, insomnia, anxiety, arrhythmias. | Irritability, headache, anxiety, weig bradycardia, difficu y concentrati |

o Cut

py

ut

morning

### Tr ea Tmen T

- Rule out medical complications; correct electrolyte ab
- Start a **benzodiazepine taper** for withdrawal sympto for hallucinations and psychotic symptoms.
- Give **multivitamins and folic acid; administer thian** (which depletes thiamine) to prevent Wernicke encepl
- Give anticonvulsants to patients with a seizure history.
- Group therapy, disulfiram, or naltrexone can aid patien
- Long-term rehabilitative therapy (eg, Alcoholic Anony

### Co mPl iCa Tio n s

- **GI bleeding from gastritis, ulcers, varices, or Mallory**
- **Pancreatitis, liver disease,** DTs, alcoholic hallucino ropathy, Wernicke encephalopathy, Korsakoff psychos drome, cardiomyopathy, anemia, aspiration pneumon ing trauma (eg, subdural hematoma).

## Eating Disorders

### ANOREXIA NERVOSA

| | |
|---|---|
| **Patients are underweight** (≥ 15% below expected weight). | Patients are of normal weight or are overweight. |
| Patients are typically not distressed by their illness and may thus be resistant to treatment. | Patients are typically distressed about their symptoms and are thus easier to treat. |
| Monitor calorie intake and weight gain; hospitalize if necessary.<br>Psychotherapy.<br>Treat comorbidities. | Psychotherapy ± antidepressants. |

weight; check BMI; check **CBC, electrolytes,** endocrine
Perform a **psychiatric evaluation** to screen patients for
s.

There ar
nervosa
- Rest
- Bing

14.
**uicide** or medical complications is > **10%**

**edical Complications of Eating Disorders**

### Treatment

See Table 2.14-13.

### Complications

See Table 2.14-14.

# Miscellaneous Disorders

## SEXUAL DISORDERS

### Sexual Changes With Aging

- Interest in sexual activity usually does not ↓ with aging
- Men usually require ↑ stimulation of the genitalia f time to reach orgasm; intensity of orgasm ↓, and the tory period before the next orgasm ↑.
- In women, estrogen levels ↓ after menopause, lead ness and thinning, which may result in discomfort du treated with hormone replacement therapy, estrogen v or other vaginal creams.

### Paraphilic Disorders

Preoccupation with or engagement in unusual sexual behaviors for > 6 months with clinically significant impa There are eight classified disorders, characterized by d (voyeurism, exhibitionism, and frotteurism), disordered philia, transvestic fetishism, fetishism), and pleasure in pain (sadism, masochism). See Table 2.14-15.

Treatment includes insight-oriented psychotherapy and

| |
|---|
| Cross-dressing for sexual arousal. |
| Touching or rubbing one's genitalia against a nonconsenting person (common in subways). |
| Sexual arousal from infli ting suffering on sexual partner. |
| Sexual arousal from being hurt, humiliated, bound, or threatened. |

sex-reassignment surgery or hormonal treatment (eg, estro-
osterone for women). Supportive psychotherapy is helpful.

ual **arousal, desire, or orgasm,** or **pain** with sexual inter-

%; one-third of cases are attributable to biologic factors
d to psychological factors.
epends on the particular condition. Pharmacologic strate-
enafil (Viagra) and bupropion (Wellbutrin). Psychothera-
include sensate focusing.

ll American adults suffer from some type of sleep disorder
he term dyssomnia describes any condition that leads to
normal rhythm or pattern of sleep. Insomnia is the most
**Risk factors** include female gender, the presence of men-
rders, substance abuse, and advanced age.

**Recomm**
**measur**
**to reest**
**sleep/w**

- Antidepressants such as SSRIs may be useful in som

## Narcolepsy

- May affect up to 0.16% of the population. Onset typica adulthood, generally before age 30. Some forms of na genetic component.
- **Dx:**
  - Manifestations include **excessive daytime somno sleep latency** on a daily basis for at least 3 month the classic symptom; patients cannot avoid falling a
  - The characteristic excessive sleepiness may be asso lowing:
    - **Cataplexy:** Sudden loss of muscle tone that lead
    - **Hypnagogic hallucinations:** Occur as the patie
    - **Hypnopompic hallucinations:** Occur as the pa
    - **Sleep paralysis:** Brief paralysis upon awakening.
- **Tx:** Treat with a regimen of **scheduled daily naps** pl such as amphetamines; give SSRIs for cataplexy.

## Sleep Apnea

- Occurs 2° to **disturbances in breathing** during sleep t **daytime somnolence** and **sleep disruption.** Etiologie tral or peripheral.
  - **Central sleep apnea (CSA):** A condition in whic respiratory effort cease. CSA is linked to **mornin** changes, and repeated awakenings during the night
  - **Obstructive sleep apnea (OSA):** A condition in w as a result of obstruction along the respiratory passa associated with **snoring.** Risk factors include **ma** prior upper airway surgeries, a deviated nasal septu tongue, and retrognathia (recession of the mandible
- In both forms, arousal results in cessation of the apneic
- Associated with **sudden death in infants and the**

## Disorder

nt with excessive thoughts, anxiety, and behaviors driven
natic symptoms that is distressing and negatively affects
occur with or without any medical illness present. High
on is often present.

uled appointments with one clinician as 1° caregiver.
ry diagnostics.

## er

mptoms or deficits of voluntary motor or sensory function
urelike movements, paralysis) incompatible with medical
nporal relationship to stress or intense emotion.

plained by other medical or neurologic causes.
gns suggesting nonorganic cause of symptoms:
Hoover sign (extension of affected leg when asked to raise
d contralateral leg) when attempting to rule out leg paral-

and resistant to opening during seizure, negative simulta-

pears with distraction.

In malin
simulate

restraining order.

- Victims of childhood abuse are more likely to becor abuse.
- **Hx/PE:**
  - Patients typically have **multiple somatic complain its,** and **unexplained injuries** with **delayed medic** may also **avoid eye contact** or act afraid or hostile.
  - Children may exhibit precocious sexual behavio **trauma, STDs,** UTIs, and psychiatric problems.
  - Other clues include a partner who answers questio refuses to leave the examination room.
- **Tx:** Perform a screening assessment of the patient's safe in her/his close personal relationships. Provide **medi support, and counseling;** educate the patient about **su** refer appropriately. **Documentation** is crucial.

## SUICIDALITY

- Accounts for 30,000 deaths per year in the United Sta all cause of death in the United States. One suicide minutes.
- Risk factors include male gender, **age > 45 years,** p (major depression, presence of psychotic symptoms), a sion to a psychiatric institution, a **previous suicide a** violent behavior, ethanol or substance abuse, recent se family suicide history (see the mnemonic **SAD PERSO**
- Women are more likely to attempt suicide, whereas m to succeed by virtue of their ↑ use of more lethal meth
- **Dx:**
  - Perform a comprehensive psychiatric evaluation.
  - Ask about family history, previous attempts, ambiva and hopelessness.
  - Ask **directly** about suicidal ideation, intent, and

ase 448
448
450
LMo NARY d ISEASE 451
se 452
E 452
454
NITIS 454
454
SYNd Ro MES 455
re 456
456
ESS SYNd Ro ME 456

Pulmonary Vascular Disease 448
PULMo NARY HYPERTENSIo N/c o R PULMo
PULMo NARY THRo MBo EMBo LISM

Neoplasms of the Lungs
LUNG No d ULES
LUNG c ANc ER

Pleural Disease
PLEURAL EFFUSIo N
PNEUMo THo RAX

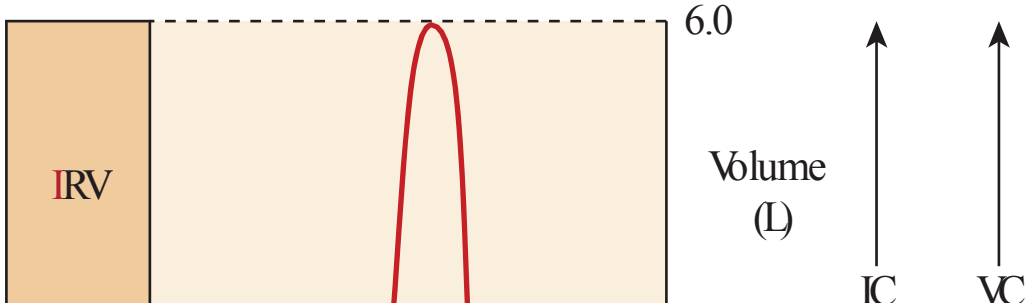
**Reversible** airway obstruction 2° to bronchial hyperreact
mation, mucous plugging, and smooth muscle hypertroph

### History/Pe

- Usually presents with dry **cough, episodic wheezin** chest tightness, often worsening at night or in early m NSAIDs can also induce asthma. (Samter's triad is asth ity, and nasal polyps.)
- **Examination: Wheezing, prolonged expiration** (↓ I **accessory muscle use,** tachypnea, tachycardia, hyper sible pulsus paradoxus.
- **Late signs:** ↓ breath sounds (ie, no wheezing), cyanos tion.

### Diagnosis

- **Spirometry/PFTs (initial diagnosis):** Essential to d pattern (↓ **$FEV_1$/FVC);** ↑ RV and total lung capacit often normal between exacerbations.
- **Methacholine challenge:** Tests for bronchial hyperre when PFTs are normal but asthma is still suspected.
- **ABGs: Initially with acute respiratory alkalosis an hypoxia.** Normalizing $P_{CO_2}$, respiratory acidosis, and r may indicate fatigue and impending respiratory f **patients are in severe respiratory distress requiring ho**
- **CXR:** Normal appearance to hyperinflation with fla phragm.



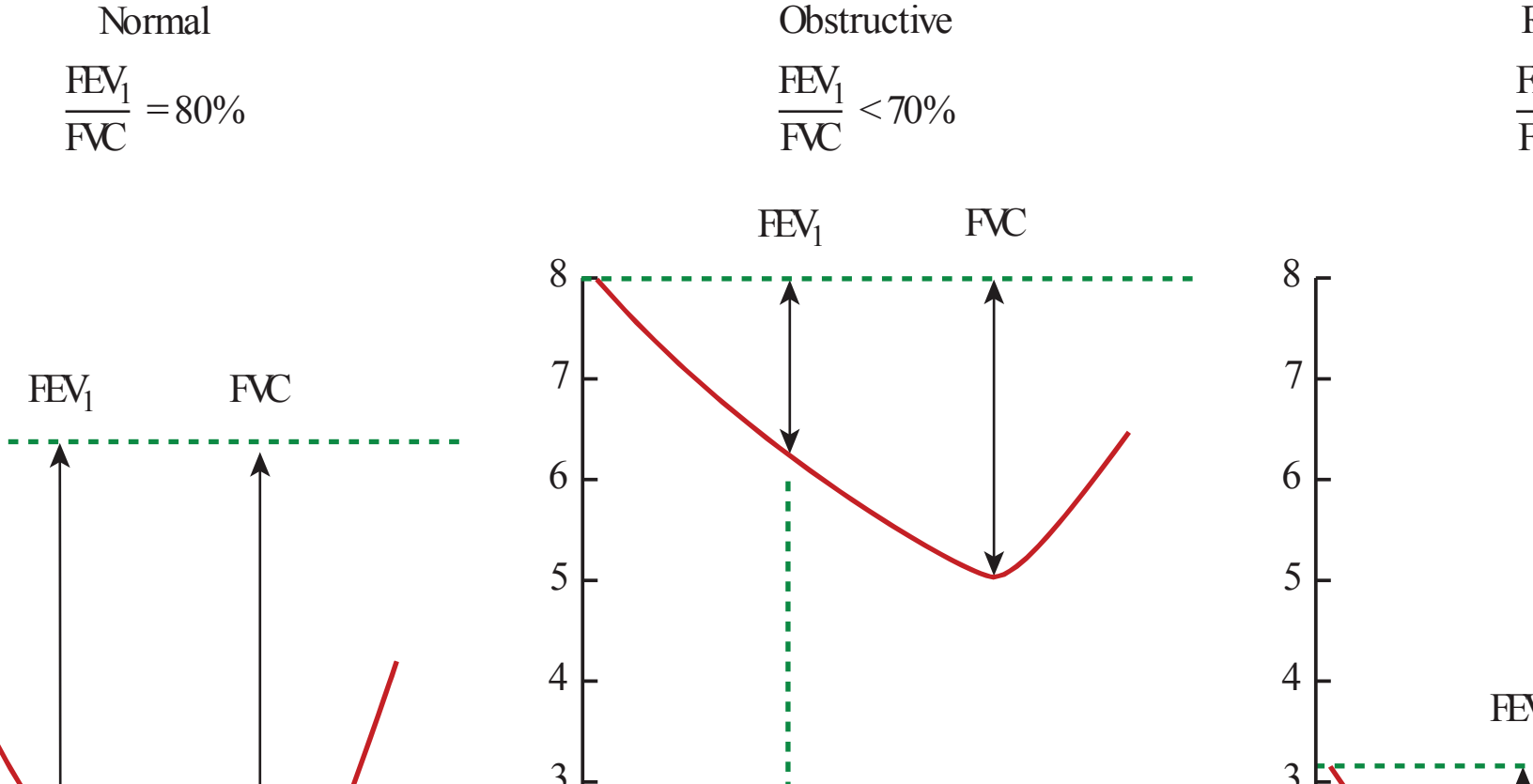
| | | | |
|---|---|---|---|
| > 80 | 60–80 | 50–60 | < 50 |
| > 75 | > 75 | > 75 | > 75 |
| 80–120 | 80–120 | 70–80 | 70 |

lergens or any potential triggers. See also Tables 2.15-2
gement.

**dilating agents** (short-acting inhaled $\beta_2$-agonists are first-; **ipratropium** (never used alone for asthma), **systemic ds,** magnesium (for severe exacerbations).
ubation in severe cases or acutely in patients with a $P_{CO_2}$ > a $P_{O_2}$ < 50 mm Hg.

Corticos
lead to t

Summar
- PRN
bron
- Long
cort
$\beta_2$-a
PO c



**eophylline:** Probably causes bronchodilation by inhibiting phosphodiesterase, thereby
MP hydrolysis and increasing cAMP levels. Usage is limited because of its narrow therap
ardiotoxicity, neurotoxicity).

events the release of vasoactive mediators from mast cells. Useful for exercise-induced b
**ective only for the prophylaxis** of asthma; not ef ective during an acute attack. Toxicit

**leuton:** A 5-lipoxygenase pathway inhibitor. Blocks conversion of arachidonic acid to leu
**ontelukast, zafi lukast:** Block leukotriene receptors.

ons—

- **Chronic:**
  - Administer long-acting inhaled **bronchodilators** a
    costeroids, systemic **corticosteroids,** cromolyn, or, r
  - Montelukast and other leukotriene antagonists a
    inhalant therapy.

## BRONCHIECTASIS

A disease caused by recurrent infection and inflammat
bronchioles that lead to fibrosis, remodeling, and **perr**
**bronchi** (see Figure 2.15-3).

**for the Chronic Treatment of Asthma**

| ms (day/ igh T) | fev$_1$ | medicaTio Ns |
|---|---|---|
| veek | ≥ 80% | No daily medications. |
| /month | | PRN short-acting bronchodilator |

s.

ronchovascular markings and **tram lines** (parallel lines out-
nchi as a result of peribronchial inflammation and fibrosis).
**CT (definitive test): Dilated airways** (ie, larger than pul-
and ballooned cysts are seen at the end of the bronchus
pes).
s a ↓ $FEV_1$/FVC ratio consistent with obstruction.

ntibiotics (oral and inhaled, for bacterial infections), con-
ticosteroids.
nopulmonary hygiene (cough control, postural drainage,
apy).
er lobectomy for localized disease or lung transplantation
.

## TIVE PULMONARY DISEASE

ng function associated with airflow obstruction. Generally
itis and/or emphysema, which are distinguished as follows:

**itis:** Productive cough for > 3 months per year for 2 con-
inical diagnosis).
estruction and dilation of structures distal to the terminal
nologic diagnosis) that may be 2° to **smoking (centrilobu-
trypsin deficiency (panlobular).**

inimal or nonspecific until the disease is advanced.
ctrum is shown in Table 2.15-4 (most patients are a com-
wo phenotypes).

**FIGURE**
Chest C
and thic
sistent w
patient.
Rx.com.)

In COPD
hyperca
may wo
mismat
respirat

Supplen
are the
improve

Treatme
exacerb
and cor
COPD e

peeted (eg, fever, productive cough, new infiltrate on C

## Treatment

See Table 2.15-5.

# Restrictive Lung Disease

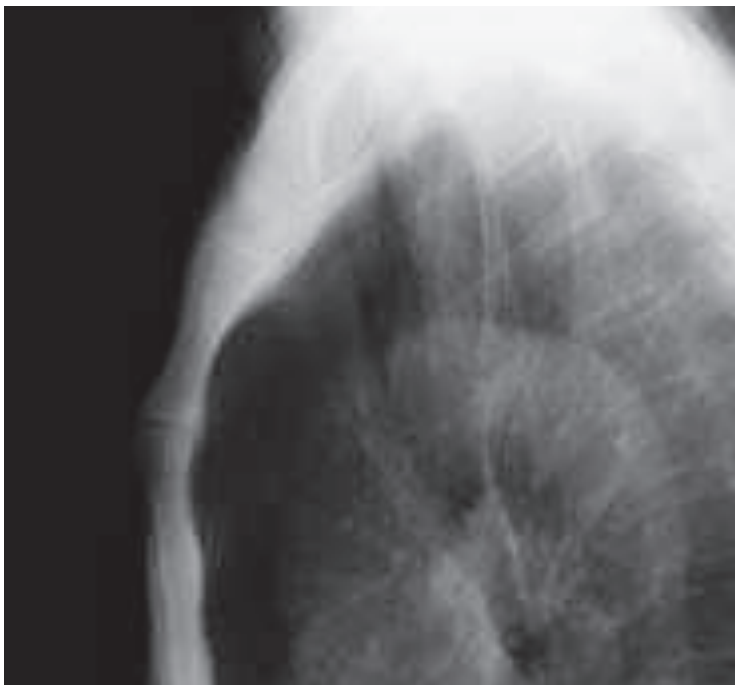
Characterized by a loss of lung compliance, restrictive lu
↑ lung stiffness and ↓ lung expansion. Table 2.15-1 and
trast obstructive with restrictive lung disease. The etiologi
disease are shown in the mnemonic **AIN'T.**

## INTERSTITIAL LUNG DISEASE

A heterogeneous group of disorders characterized by **in**
**fibrosis** of the interstitium.

- Advanced disease: Cystic spaces develop in the lung
  combing").





| | | |
|---|---|---|
| **biotics.** | (if severe hypoxemia/ hypercapnia, respiratory fatigue, changes in mental status). | |
| led $\beta_2$**-agonists,** **cholinergics** ropium), systemic or led **corticosteroids.** | Give **supplemental** $\mathbf{O_2}$ if patient has (1) hypoxemia (2) right heart failure, or (3) polycythemia. | Smoking cessation, pneumococcal and flu accines. |

**Exposure related** (eg, "coal worker's pneumoconiosis," sis, berylliosis, hypersensitivity pneumonitis, radiation- (2) **ILD associated with systemic disease or connective** (polymyositis/dermatomyositis, sarcoidosis, amyloidosis, tomyositis, CREST syndrome); (3) **idiopathic** (idiopathic sis, cryptogenic organizing pneumonia, acute interstitial

**hallow, rapid breathing;** dyspnea with exertion; and a uctive cough.
ave cyanosis, inspiratory squeaks, fine or "Velcro-like" g, or right heart failure.

**l test):** Reticular, nodular, or ground-glass pattern; **"honey-** evere disease).
**FVC, ↓ $\mathbf{DL_{CO}}$ (initial test):** May be normal if the cause y, **normal $\mathbf{FEV_1/FVC}$.** Serum markers of connective tis- ld be obtained if clinically indicated.

Medicat
cause o
disease
nitrofura
and long
(eg, PaC



FIGUR

plaques), nervous system, heart, and kidney may be all

### Diagnosis

- **CXR/CT (initial test):** Radiographic findings of **lym nodules** are used to stage the disease.
- **Biopsy (definitive test):** Lymph node biopsy or t assisted thoracoscopic lung biopsy reveals **noncaseatin**
- **PFTs:** Restrictive or obstructive pattern and ↓ diffusion
- **Other findings:** ↑ **serum ACE levels** (neither sen **hypercalcemia,** hypercalciuria, ↑ alkaline phosphatase ment), lymphopenia, cranial nerve defects, arrhythmia

### Treatment

Systemic **corticosteroids** are indicated for deteriorating constitutional symptoms, hypercalcemia, or extrathoracic

## HYPERSENSITIVITY PNEUMONITIS

Alveolar thickening and noncaseating granulomas 2° to e sure (eg, mold, hot tubs, cockatiels, down feather antigens

### History/Pe

- **Acute:** Dyspnea, fever, malaise, shivering, and cough after exposure. Gather a job/travel history to determine
- **Chronic:** Patients present with progressive dyspnea; fine bilateral rales.

### Diagnosis

The appearance on CXR/CT is variable, but upper lobe f feature of chronic disease.

### Treatment

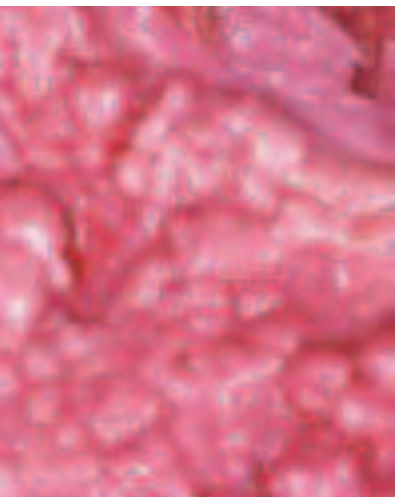
| | | |
|---|---|---|
| | upper lung zones. | |
| k in mines or quarries or with glass, ery, or silica. | Small (< 1-cm) nodular opacities in upper lung zones; **eggshell calcifi ations.** | ↑ **risk** Progre |
| k in high-technology fields such as **space, nuclear, and electronics ts;** ceramics industries; foundries; ng facilities; dental material sites; dye manufacturing. | Diffuse infil ates; hilar adenopathy. | **Requi treatm** |

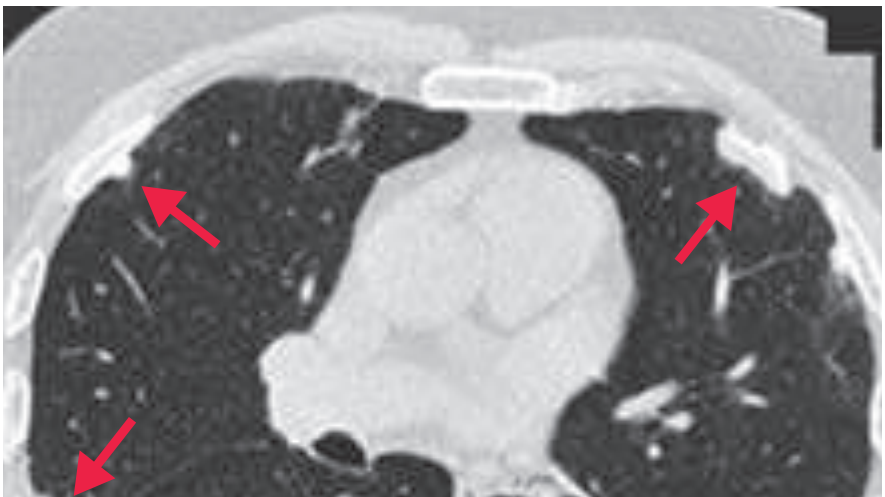
with restrictive disease.

ortive therapy and supplemental $O_2$.

## MONARY SYNDROMES

disorders characterized by eosinophilic pulmonary infil- peripheral blood eosinophilia. Includes allergic broncho-







### Treatment

Removal of the extrinsic cause or treatment of underlyin
minths) if identified. Corticosteroid treatment may be use

# Acute Respiratory Failure

## HYPOXEMIA

↓ $P_{O_2}$; causes include **ventilation-perfusion (V/Q) mis
shunt, hypoventilation,** low inspired $O_2$ content (importa
**diffusion impairment.**

### History/Pe

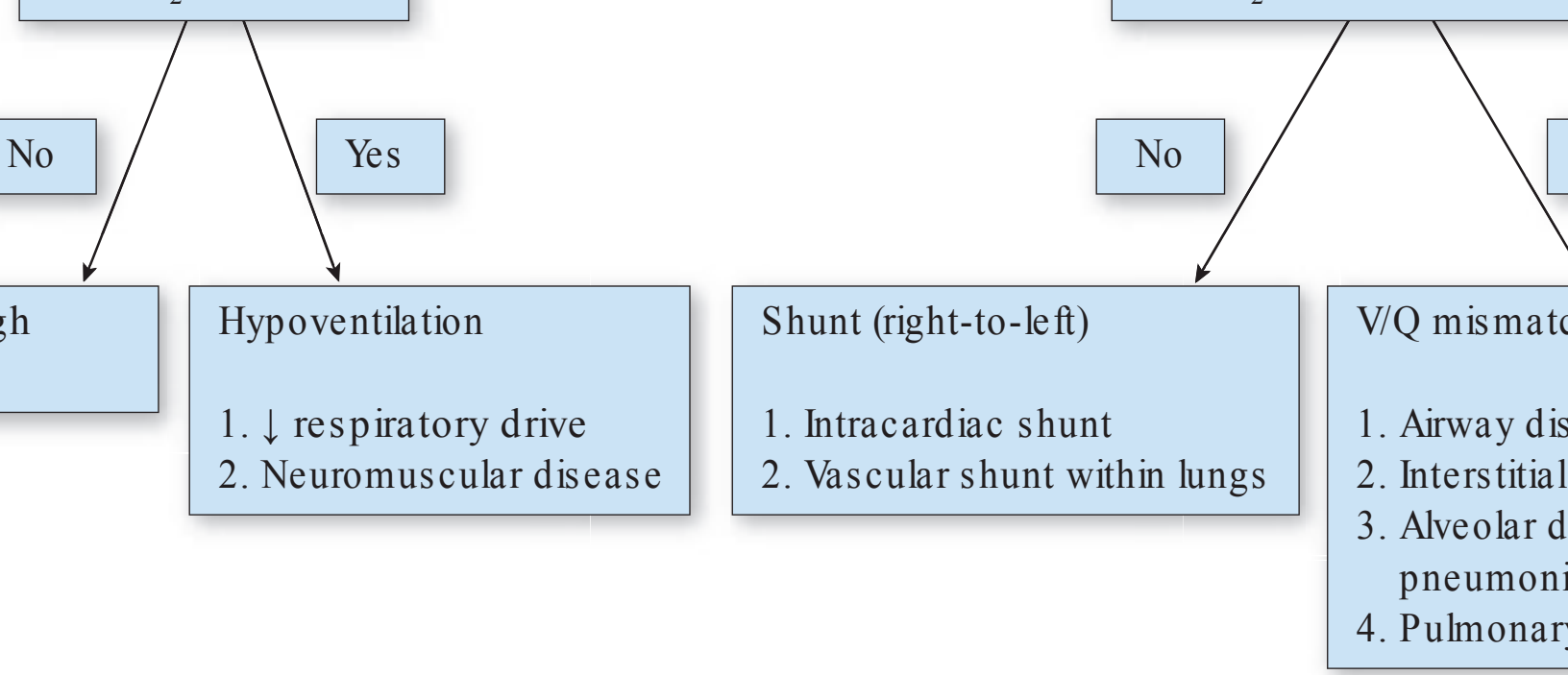
Findings depend on the etiology. ↓ **$SpO_2$ saturation,** c
shortness of breath, pleuritic chest pain (caused by wheez
altered mental status may be seen.

### Diagnosis

- **ABGs (initial test):** Calculate the **alveolar-arterial (A-**
  $([P_{atm} - 47] \times FiO_2 - [PaCO_2/0.8]) - PaO_2$.
- **Pulse oximetry:** Demonstrates ↓ **$SpO_2$** saturation.
- **CXR:** To evaluate for an infiltrative process (eg, pneur
  large pleural effusion, or pneumothorax and to assess
  gradient suggests shunt, **V/Q mismatch,** or **diffusion**
  2.15-7 summarizes the approach toward hypoxemic pa

### Treatment

- Address the underlying etiology.
- Administer $O_2$ before initiating evaluation.
- ↑ oxygenation parameters if the patient is on mechan




**termination of the mechanism of hypoxia.**

**onset** (12–48 hours) tachypnea, dyspnea, and tachycardia abored breathing, diffuse high-pitched rales, and hypox- of one of the systemic inflammatory causes or exposure. are as follows:

**njury):** Normal physical examination; possible respiratory

**ours):** Hyperventilation, hypocapnia, widening A-a gradient. respiratory failure, tachypnea, dyspnea, ↓ lung compli- ales, diffuse chest opacity on CXR (see Figure 2.15-8). hypoxemia unresponsive to therapy; ↑ intrapulmonary olic and respiratory acidosis.



end-organ damage.

- Mechanical ventilation with low tidal volumes (4–6 weight) to minimize ventilator-induced lung injury.
- Use PEEP to recruit collapsed alveoli, and titrate achieve adequate oxygenation.
- Goal oxygenation is $PaO_2$ > 55 mm Hg or **$SaO_2$ > 88%**
- Slowly wean patients from ventilation, and follow w (see Table 2.15-8).

# Pulmonary Vascular Disease

## PULMONARY HYPERTENSION/COR PULMONALE

Cor pulmonale is right-sided heart failure due to pulmona

A mean pulmonary arterial pressure of > 25 mm Hg (no Five classification categories were defined in 2008 with re

- Pulmonary arterial hypertension.
- ↑ pulmonary venous pressure from left-sided **heart dis**

**TABLE 2.15-8. Criteria for Extubation from Mechanical Ventilati**

| Parameter | Va |
|---|---|
| **Pulmonary mechanics** | |
| Frequency/TV ratio (most predictive of successful extubation) | < 105 |
| Vital capacity | > 10–15 |
| Resting minute ventilation (TV × rate) | > 10 L/m |
| Spontaneous respiratory rate | < 33 brea |

e. Patient may also be hypoxemic, especially on exertion.

rgement of central pulmonary arteries.
tes RVH.
(initial test) and right heart catheterization (definitive
igns of right ventricular overload and may aid in the diag-
erlying cause.

anticoagulation, vasodilators, and diuretics if symptoms
are present. Treat underlying causes of 2° pulmonary
treatments for pulmonary arterial hypertension include
elin receptor antagonists, and phosphodiesterase inhibi-
have vasoreactivity and respond well to high-dose calcium

## MBOEMBOLISM

pulmonary vasculature by a blood clot. **Ninety-five per-
ginate from DVTs** in the deep leg veins. Often leads to
n, right heart failure, and hypoxemia.

sing to thromboembolism are summarized by Virchow
2.15-9).
**dden-onset or subacute dyspnea, pleuritic chest pain,**
cough, tachypnea, tachycardia, and, rarely, hemoptysis.

**how Triad for Venous Thrombosis**

Other et
include
fluid em
cardiac
endovas
emboli).

Q

A 25-y
intuba
asthm
is sent
a pH

- **ABGs:** Respiratory alkalosis (2° hyperventilation, $P_{O_2}$ <
- **CXR:** May show atelectasis, pleural effusion, **Hampt** shaped infarct), Westermark sign (oligemia/collapse to PE).
- **ECG:** Most commonly reveals **sinus tachycardia.** **S1Q3T3 is rare**—acute right heart strain with an S wave in lead III, and an inverted T wave in lead III.
- **V/Q scan:** May reveal areas of mismatch to predict lo high probability of PE; use if contraindications to CT indication to contrast], pregnancy [contraindication t tive for PE, but not specific, especially if there is under
- **Lower extremity venous ultrasound:** Can detect a given off PE, not used to diagnose the PE. Specific an specific but not sensitive for PE.

### Treatment

- **Anticoagulation:**
  - **Acute:** Heparin (bolus then heparin infusion or lo heparin [LMWH] subcutaneously).
  - **Chronic:** Anticoagulation for 3–6 months or du (goal INR = 2–3).
- **IVC filter:** Indicated in patients with a documented lo if anticoagulation is contraindicated or if patients e emboli while anticoagulated.
- **Thrombolysis:** Indicated in cases of massive PE causin and hemodynamic instability.



atment (see Table 2.15-10).

c, or patients may present with chronic cough, dyspnea, eath. Always inquire about smoking and exposure history, l with greater cancer risk.

**on** is indicated for nodules at high risk for malignancy. s can be followed with serial **CXR or CT.**
nostic procedure is indicated if the size of the nodule ↑.

of **cancer death** in the United States. Risk factors include ept for bronchoalveolar carcinoma) and radon or asbestos as follows (see also Table 2.15-11):

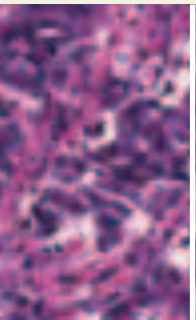
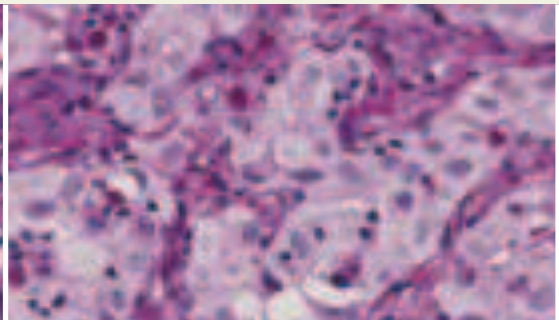
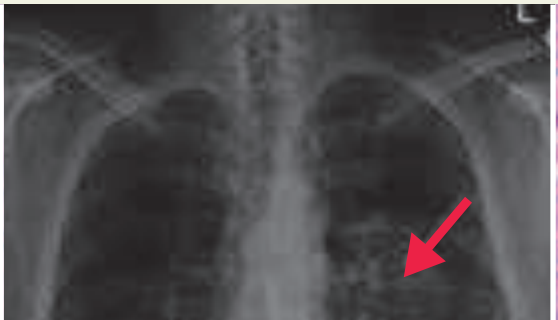
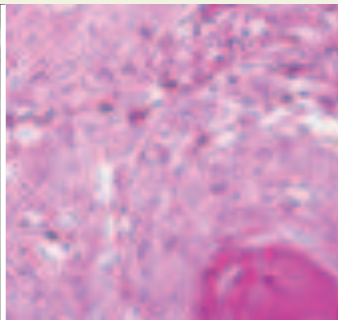
**cancer (SCLC):**
ated with **cigarette exposure.** Central location (see Fig- **neuroendocrine origin;** associated with paraneoplastic ee Table 2.15-12).
e often found on presentation in intrathoracic and extra- such as brain, liver, and bone.
**lung cancer (NSCLC):** Group of cancers, with the most eing adenocarcinoma, squamous cell carcinoma (SCC), arcinoma. These cancers are less likely than SCLC to early stage.

**aracteristics of Benign and Malignant Lung Nodules**

Lung no
- Rec
- Fron Stat
- Fron histo

Squamo
Sentral

| | | |
|---|---|---|
| | common. Inoperable; treat with chemotherapy. | Chro |
| | **Non–Small Cell** | |
| ripheral | Most common lung cancer in nonsmokers and overall (except for metastases). Activating mutations include KRAS, EGFR, and ALK. Associated with hypertrophic osteoarthropathy (clubbing).<br>Bronchioloalveolar subtype (adenocarcinoma in situ): CXR often shows hazy infiltrates similar to pneumonia; excellent prognosis. | Glan his mu be<br>Bron gr se "th wa |
| entral | Hilar mass arising from bronchus (see red arrow in Figure C below); Cavitation; Cigarettes; hyperCalcemia (produces PTHrP).v | Kerat in Fig inter |
| ripheral | Highly anaplastic undifferentiated tumor; poor prognosis. Less responsive to chemotherapy; removed surgically. | Pleor Can s |
| - | Excellent prognosis; metastasis rare.<br>Symptoms usually due to mass effect; occasionally carcinoid syndrome (5-HT secretion → flushin , diarrhea, wheezing). | Nest cells; |



B

**small cell lung cancer.** Note the central location of the tumor in the PA **(A)** and later
D Anderson Manual of Medical Oncology. New York, NY: McGraw-Hill; 2006.)

**oma:**
mon lung cancer; **peripheral** location.
**bronchoalveolar carcinoma,** associated with multiple nterstitial infiltration, and prolific sputum production but **ociated with smoking.**

**raneoplastic Syndromes of Lung Cancer**

| Syndrome | Histologic Type |
|---|---|
| ushing syndrome (ACTH) | Small cell |
| ADH leading to hyponatremia | Small cell |
| ypercalcemia (PTHrP) | Squamous cell |
| ynecomastia | Large cell |
| ypertrophic pulmonary osteoarthropathy ncluding digital clubbing) | Non–small cell |
| eripheral neuropathy | Small cell |
| ubacute cerebellar degeneration | Small cell |
| yasthenia (Lambert-Eaton syndrome) | Small cell |
| ermatomyositis | All |

Lung ca
in LABE

**L**iver
**A**drenals
**B**rain
**B**one



rome.
yn-
VC by a
ced with
Emer-
e, 7th ed.

- **SVC syndrome:** Obstruction of the SVC with sup
  engorgement and facial swelling (see Figure 2.15-11).
- **Hoarseness:** 2° to recurrent laryngeal nerve involveme
- Many **paraneoplastic syndromes** (see Table 2.15-12).

### Diagnosis

- CXR or chest CT (initial test).
- Fine-needle aspiration (CT guided) for peripheral les
  (biopsy or brushing) for central lesions. These are the **d**

### Treatment

- **SCLC: Unresectable.** Often responds to radiation and
  tially but usually recurs; has a low median survival rate
- **NSCLC: Surgical resection** in early stages. Suppleme
  ation or chemotherapy (depending on the stage). Pall
  or chemotherapy is appropriate for symptomatic but un

## Pleural Disease

### PLEURAL EFFUSION

An abnormal **accumulation of fluid in the pleural spac**
lows:

- **Transudate:** 2° to ↑ pulmonary capillary wedge p
  ↓ oncotic pressure.
- **Exudate:** 2° to ↑ pleural vascular permeability.

Table 2.15-13 lists the possible causes of both transudates

**TABLE 2.15-13. Causes of Pleural Effusion**

an exudate if it meets **any of Light's criteria** (see Table

the underlying condition causing the effusion.
rapneumonic effusions and empyemas require **chest tube**
tion to **antibiotic therapy** or **pleurodesis.**

Complic
necessit

the pleural space that can lead to pulmonary collapse.

trating trauma, infection, and positive-pressure mechani-
ubtypes are as follows:
**pneumothorax:** 2° to rupture of subpleural apical blebs
**tall, thin young men**).
**ax:** 2° to COPD, trauma, infections (TB, Pneumocystis
rogenic factors (thoracentesis, subclavian line placement,
mechanical ventilation, bronchoscopy).
**nothorax:** A pulmonary or chest wall defect acts as a
**causing air trapping in the pleural space.** Buildup of
ediastinum to the **opposite** side of the chest, which can
return to the heart, leading to **cardiac arrest** and **hemody-**
**y.** Shock and death unless immediately treated.

Present

**P-THO**

**P**leuritic
**T**racheal
**H**yperres
**O**nset su
**R**educed
**A**bsent f
**X**-ray sho

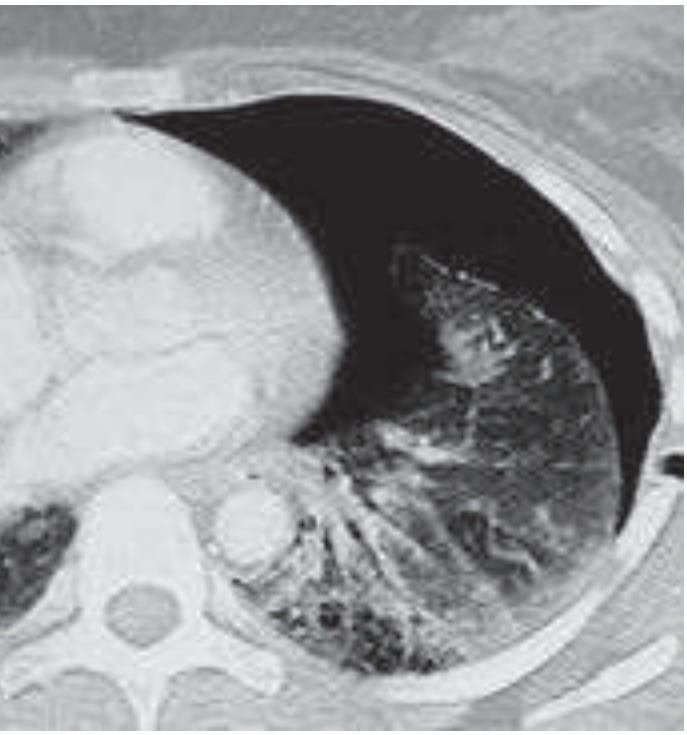
ute onset of unilateral pleuritic chest pain and dyspnea.
veals tachypnea, diminished or absent breath sounds,
↓ tactile fremitus, and JVD 2° to compression of the

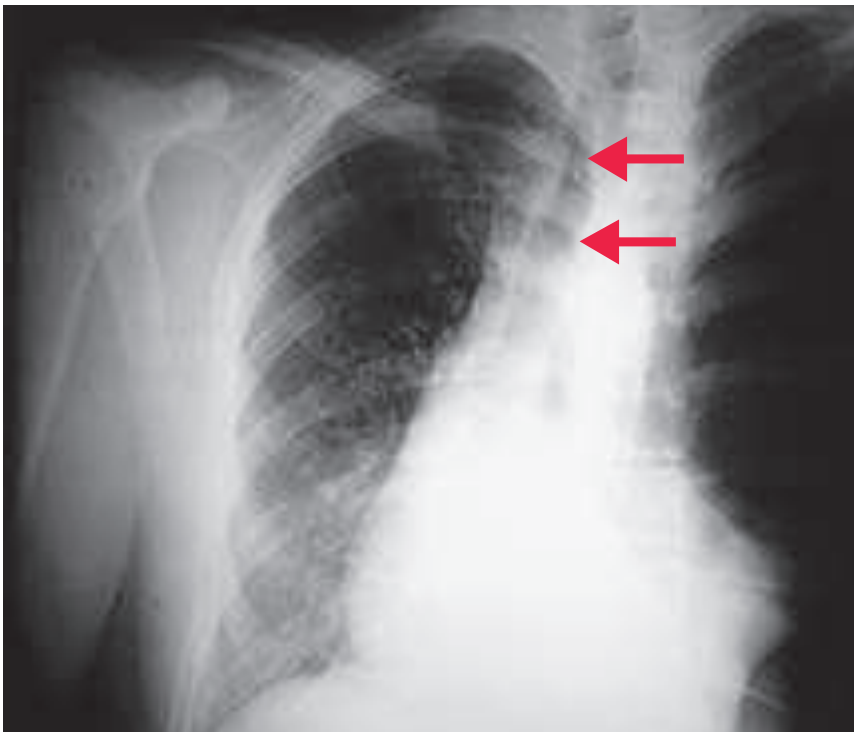
othorax also presents with respiratory distress, hypoxia, **tra-**
**and hemodynamic instability.**

thorax
first,

ond intercostal space at the midclavicular line) follo
placement.

- Small pneumothoraces may resorb spontaneously. S
helpful.
- Large, symptomatic pneumothoraces require chest tub





B

**horax. (A)** Pneumothorax. CT shows collapsed left lung. **(B)** Tension pneumothora
diaphragm (below the field of view) and rightward mediastinal/tracheal shift. (Reproduc
York, NY: McGraw-Hill; 2015.)

468 Nephrolithiasis
468 Polycystic Kidney Disease
469
470 Hydronephrosis
471 Scrotal Swelling
472
473 Erectile Dysfunction
473 Benign Prostatic Hyperplasia
474 Prostate Cancer
474 Bladder Cancer
474 Renal Cell Carcinoma
475 Testicular Cancer
477
477
477
477
478

ce water
can

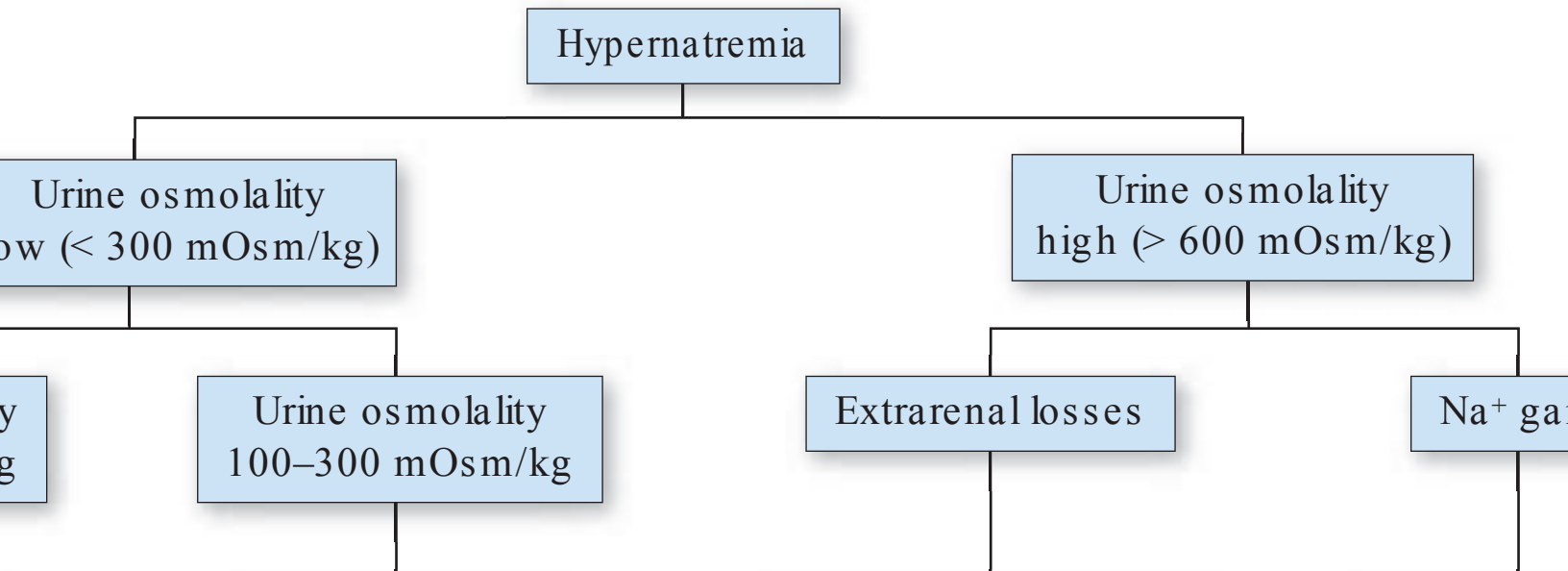
Presents with **thirst** (due to hypertonicity), **neurologic s** mental status changes, weakness, focal neurologic deficit seizures.

## Diagnosis

See Figure 2.16-1 for the hypernatremia diagnostic al based on **urine osmolality and urine sodium.**

## Treatment

- Determine volume status. If hypovolemic **with unsta** **isotonic 0.9% NaCl** before correcting free water de and asymptomatic, can treat with $D_5W$.
- Determine free water deficit. **Water deficit** = Total b Na / 140] – 1).
  - Total body water (TBW) is ~ 60% of lean body weig
  - Replace with **$D_5W$,** 0.45% NaCl, or enteral water.
- **Determine rate of replacement.** Correction of chr (> 48 hours) should be accomplished **gradually over** mEq/L/hr) to prevent neurologic damage 2° to cerebra
- Euvolemic and hypervolemic hypernatremia are rare combination of diuretics and $D_5W$ to remove excess N

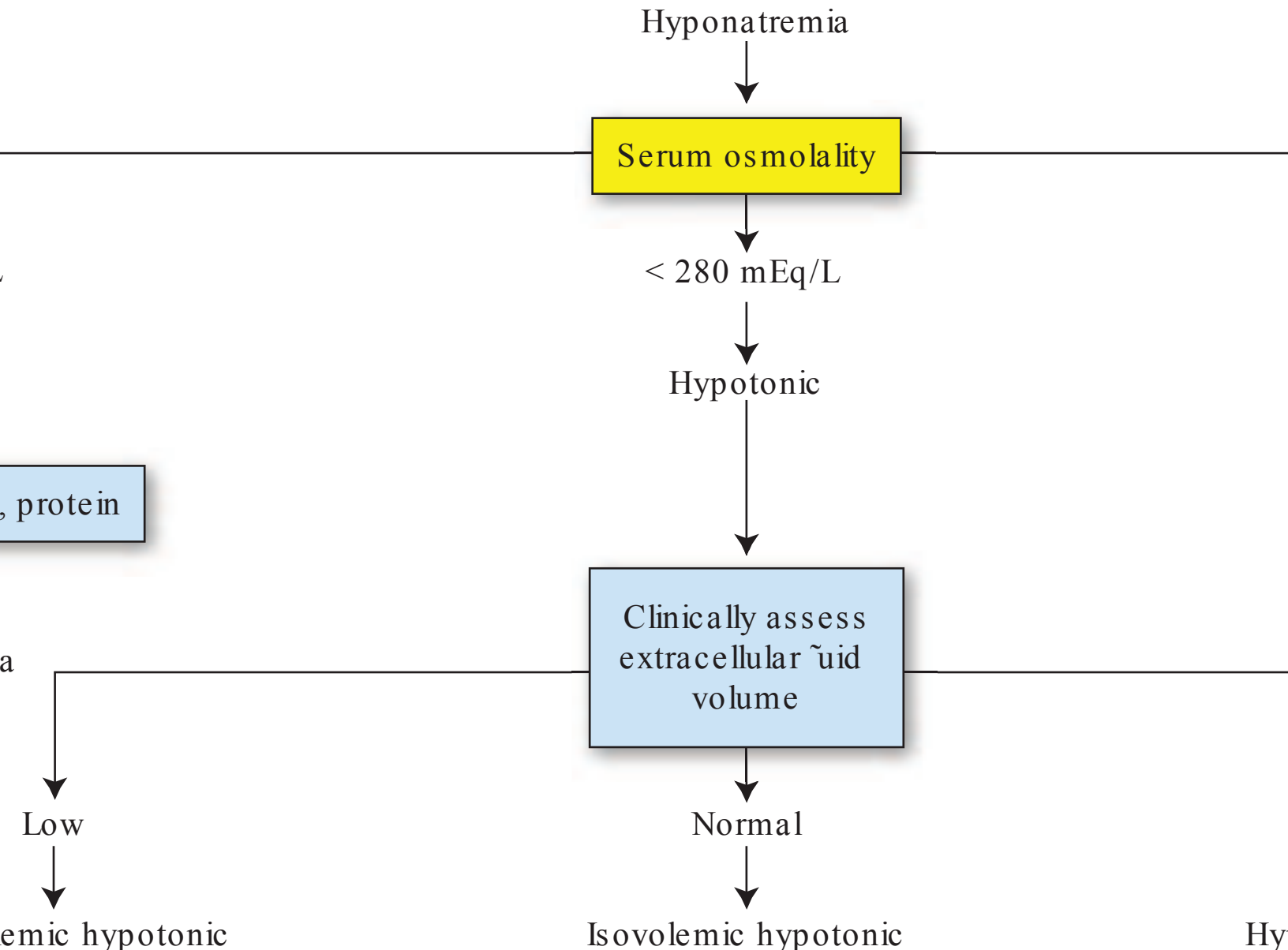


**smolality ($S_{osm}$) as the first step in evaluation: $S_{osm}$ = (2 UN/2.8) + (glucose/18). Hypotonic hyponatremia is the d is further categorized by volume status.** See Figure agnostic algorithm.

g disorder. Treat hypervolemic and euvolemic etiologies **ction** ± diuretics. If **hypovolemic,** replete volume with **NS.** remia (Na < 120 mEq/L), consider hypertonic saline. tremia (> 72 hours' duration) should be corrected slowly y) in order to prevent central pontine myelinolysis or nation (symptoms include paraparesis/quadriparesis, dys- a). See Figure 2.16-3.

hyponat
< 120 m
best rep

Correcti
rapidly c
**myelino**



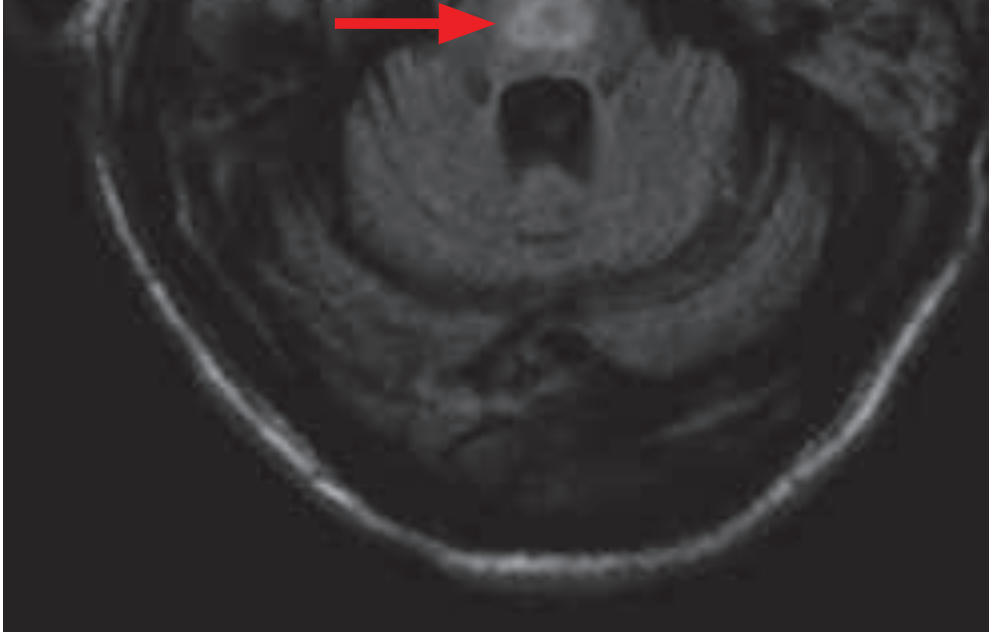


**FIGURE 2.16-3. Central pontine myelinolysis on MRI.** (Used with pe
Gaillard.)

## HYPERKALEMIA

Serum potassium > 5 mEq/L. Etiologies are as follows:

- **Spurious:** Hemolysis of blood samples, fist clenching
  delays in sample analysis, extreme leukocytosis or thro
- ↓ **excretion:** Renal insufficiency, drugs (eg, spironol
  amiloride, ACEIs, trimethoprim, NSAIDs), hypoald
  renal tubular acidosis (RTA), calcineurin inhibitors.
- **Cellular shifts:** Cell lysis, tissue injury **(rhabdomyoly**
  **drome,** insulin deficiency, acidosis, drugs (eg, succi
  arginine, β**-blockers**), exercise, resorption of blood (he
  ing).
- **Increase intake:** Food (most fruits, potatoes), iatrogeni

### History/Pe

May be asymptomatic or may present with nausea, vomiti
**areflexia, weakness,** flaccid paralysis, arrhythmias, and par

eatic transplants (can cause bowel necrosis).
an be used for increasing urinary excretion of potassium.
priate for patients with renal failure or for severe, refrac-

3.6 mEq/L. Etiologies are as follows:

**ifts:** Insulin, $\beta_2$-agonists, and alkalosis all cause potassium
larly (see Figure 2.16-5).
hea, chronic laxative abuse, vomiting, NG suction.
**uretics** (eg, loop or thiazide), 1° mineralocorticoid excess
teronism, ↓ circulating volume, Bartter and Gitelman syn-
eg, gentamicin, amphotericin), DKA, **hypomagnesemia,**
RTA, polyuria.

e, **muscle weakness or cramps, ileus,** hyporeflexia, pares-
ysis, and ascending paralysis.

n levels.
renal from GI losses with 24-hour or spot urine potas-

**CG** may show **T-wave flattening, U waves** (an additional
wave), and ST-segment depression, leading to AV block
cardiac arrest. See Figure 2.16-6.

Patients
hypokal
have on
- Surr
- Diur
- Bart
in th
- Gite
defe
tubu

Hypoka
GI losse

If a patie
levels m
Hypoka
digitalis
compet
$K^+$ pump

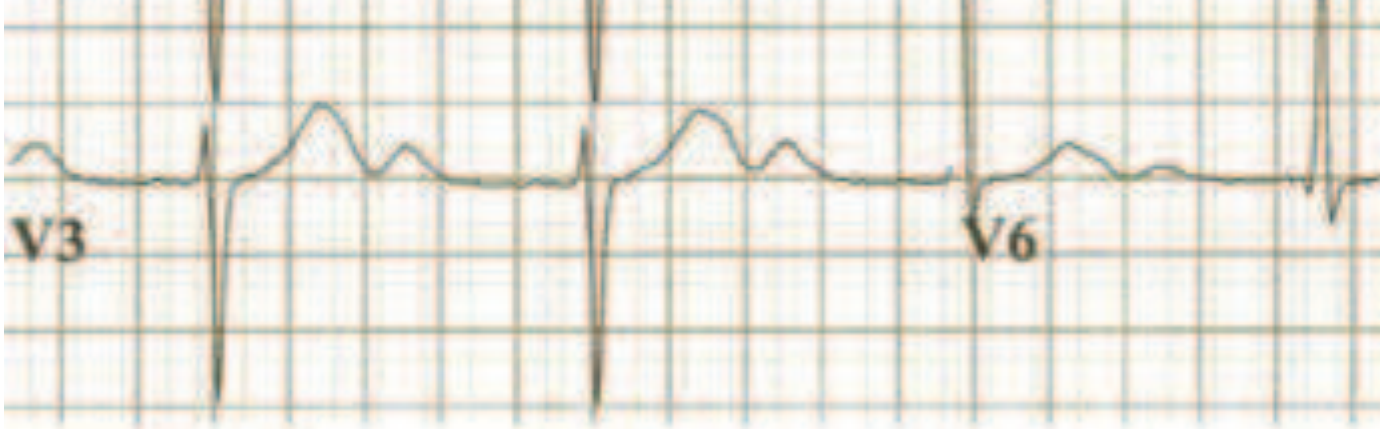



**FIGURE 2.16-6. Hypokalemia on ECG.** Prominent U wave indicat
with permission from Van Beers EJ, Stam J, van den Bergh WM. Licorice consumption as a cau
encephalopathy syndrome: a case report. Crit Care. 2011;15(1):R64.)

### Treatment

- Treat the underlying disorder.
- Oral and/or IV **potassium repletion.** Do not exceed 20
- Replace **magnesium,** as this deficiency makes potass
  difficult.

## HYPERCALCEMIA

Serum calcium > 10.2 mg/dL (corrected for serum album
mon causes are **hyperparathyroidism and malignancy**
squamous cell carcinoma, multiple myeloma). Other cau
in the mnemonic **CHIMPANZEES.**

### History/Pe

Usually asymptomatic but can present with **bones** (ost
**stones** (kidney stones), abdominal **groans** (anorexia, con
**chiatric overtones** (weakness, fatigue, altered mental statu

### Diagnosis

- Order a total/ionized calcium, albumin, phosphate, PT

**as (classic symptoms in the transient hypocalcemia in**
**),** and convulsions.
cited from tapping of the facial nerve **(Chvostek sign)** and
er arterial occlusion by a BP cuff **(Trousseau sign)** are
hat are most commonly seen in severe hypocalcemia.

**nd PTH** are the most useful tests in the diagnosis. See
hapter for interpretation of PTH levels.
s had a thyroidectomy, review the operative note to deter-
s any potential damage to the parathyroid glands.
induce PTH resistance), albumin, 25-OH vitamin D, and
D levels may also be helpful depending on clinical situa-

CG may show a **prolonged QT interval.**

ying disorder.
etion.
**calcium supplements;** give oral and IV calcium for severe

< 1.5 mEq/L. Etiologies are as follows:

trition, malabsorption, short bowel syndrome, TPN, PPIs.
, diarrhea, vomiting, hypercalcemia, **alcoholism.**
**DKA,** pancreatitis, extracellular fluid volume expansion.

ptoms may include hyperactive reflexes, tetany, paresthe-

Serum c
low in h
calcium
$Ca^{2+}$ + 0

Alcohol
patient
hypoma

ASA (sal
both a r
respirato

Specifi
causes

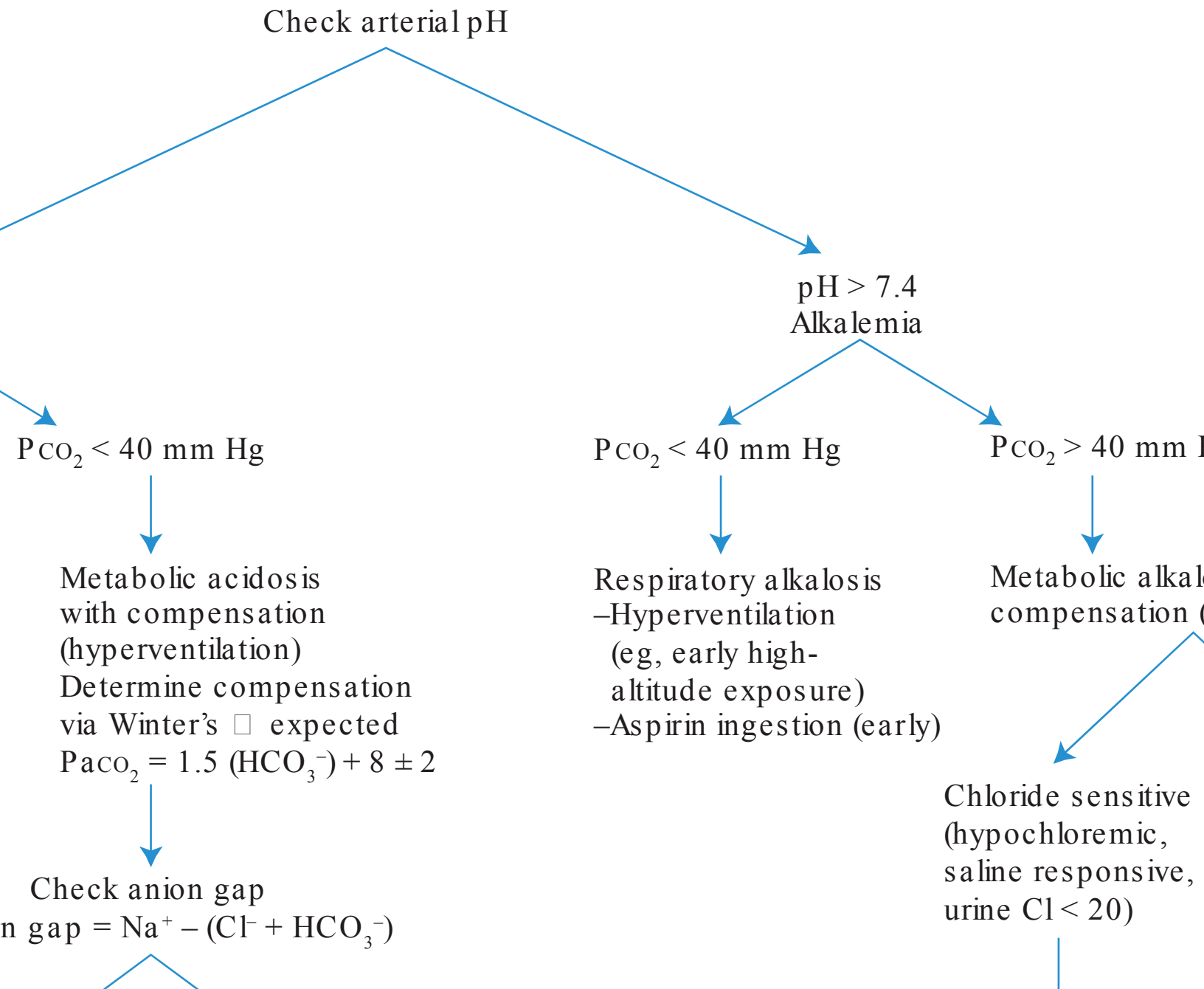
**MUDP**

**IV (distal)** is the **most common form** (see Table 2.16-1).

# Acute Kidney Injury

Formerly known as **acute renal failure.** Defined as an abr
tion leading to the retention of creatinine and BUN. ↓ ur
defined as < 500 mL/day) is not required for acute kidne
tions include metabolic acidosis, electrolyte abnormalitie
load; ultimately may progress to CKD. See Table 2.16-2 fo

story
f ATN.



| | | |
|---|---|---|
| hypercalciuria, ifosfamide. | Fanconi, cystinosis, ifosfamide. | (eg, rena inhibitio |
| Replace bicarbonate. | Thiazides, volume depletion to increase reabsorption of bicarbonate. | Furosem replacer |
| **Nephrolithiasis.** | Rickets, osteomalacia. | |

## Kidney Disease

**nths** of GFR < 60 mL/min **or** kidney damage (structural/ lities). Most commonly due to **diabetes mellitus (DM),** glomerulonephritis. Another commonly tested etiology is sease (PKD).

natic until GFR is < 30 mL/min, but patients gradually s and symptoms of uremia (anorexia, nausea, vomiting, "uremic frost," delirium, seizures, coma).

derangements include the following:

N and creatinine).
hypertension, edema, CHF, pulmonary edema).
sis.

ropoietin production).
stasis caused by impaired platelet aggregation.
yperphosphatemia, 2° hyperparathyroidism (↓ phosphate



Indicat

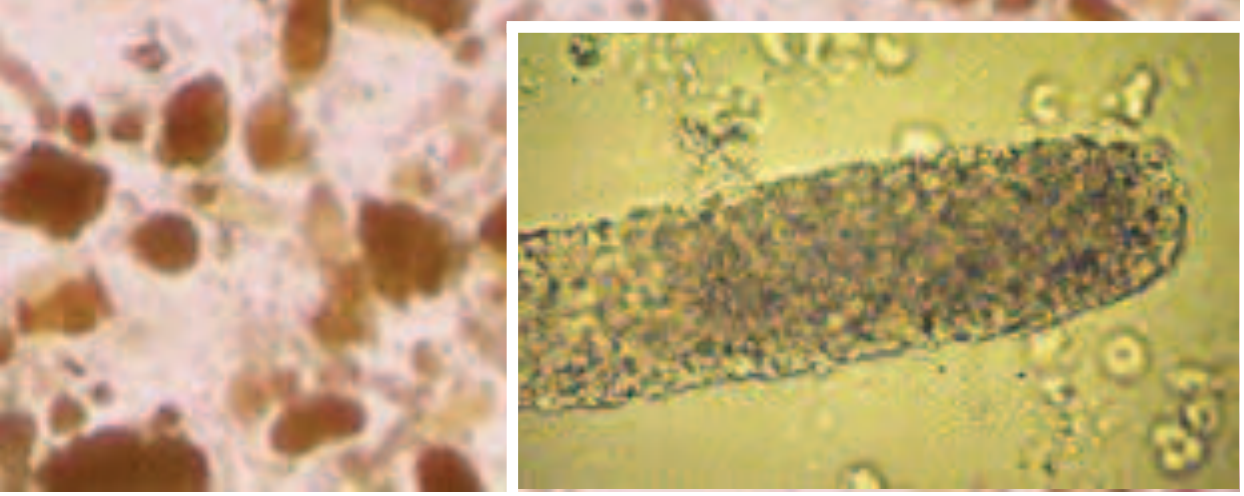
**AEIOU**

**A**cidosis
**E**lectroly
**I**ngestion
metha
ethyle
**O**verload
**U**remic s
encep
prurit



Do not g
patients
failure, b
metabo

| | | |
|---|---|---|
| s of hypovolemia<br>dia, hypotension) or other<br>g disease process (liver<br>phrotic syndrome). | Hx of drug exposure (aminoglycosides, NSAIDs, penicillins), **contrast media.** Creatinine spikes in **5–10 days** with drug injuries, **24 hours** with contrast injuries. | Decreased uri<br>pain, distende<br>shows **postvo** |
| | **lab Values** | |
| | < 15:1 | Varies. |
| | > 2% | > 1–2% |
| /L | > 40 mEq/L | > 40 mEq/L |
| sm/kg | < 350 mOsm/kg (isosthenuria, damaged tubules cannot reabsorb water). | < 350 mOsm/k |
| asts (normal findin , but<br>ne depletion). | RBC casts/dysmorphic RBCs (glomerulonephritis), WBCs/ **eosinophils** (AIN), "muddy-brown cast" (ATN) (see Figure 2.16-8), WBC casts (pyelonephritis), fatty casts (nephrotic syndrome). | |
| oid nephrotoxic drugs | Prevent contrast nephropathy with | Bladder cathet |



**ndings on microscopic urine examination in AKI.** "Muddy brown
nission from Dr. Serban Nicolescu.)

arizes the mechanisms of action and side effects of com-
s. Figure 2.16-9 provides a review of nephron physiology
f action.

## lar Disease

OME

erular inflammation, also called glomerulonephritis. Pro-
sent but is usually < 1.5 g/day. Causes are summarized in

are oliguria, macroscopic/microscopic hematuria (tea- or
hypertension, and **edema (can also present with pulmo-**

Postinfe
present
and has
present
and has

Granulo
kidne
Microsc
lung.
Churg-S

| | | | |
|---|---|---|---|
| nide, ethacrynic<br>metanide,<br>de. | Ascending loop of Henle. | Inhibit $Na^+/K^+/2Cl^-$ transporter. | Wa<br>alk<br>↓ M<br>alle<br>aci |
| hlorothiazide. | Distal convoluted tubule. | Inhibit $Na^+/Cl^-$ transporter. | Wa<br>alk<br>↑ g<br>↑ u<br>par |
| lactone,<br>rene, amiloride. | Cortical collecting tubule. | Aldosterone receptor antagonist (spironolactone); block sodium channel (triamterene, amiloride). | Me<br>ant<br>inc<br>(sp |

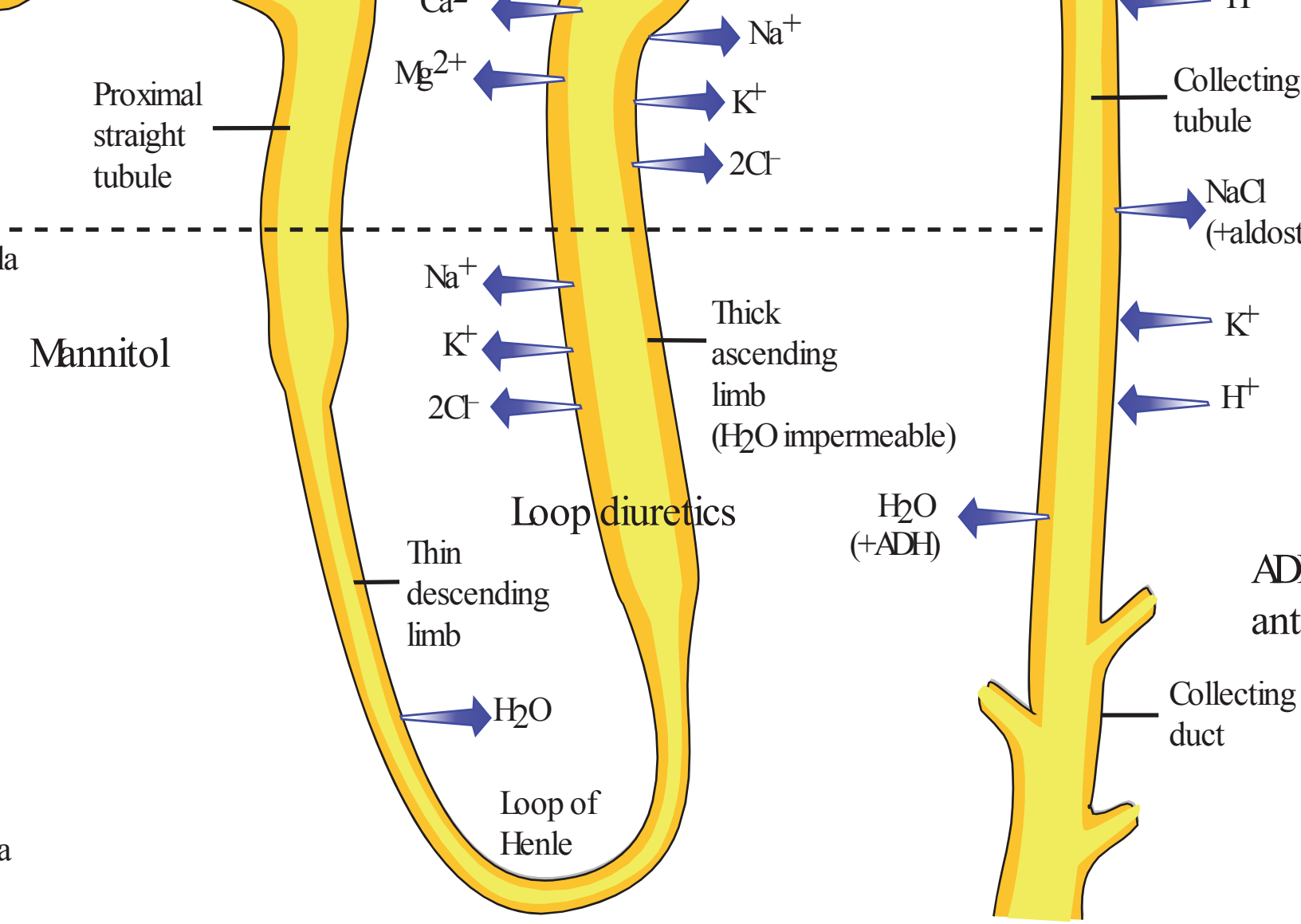
### Treatment

- Treat hypertension, fluid overload, and uremia with sa
  tion, diuretics, and, if necessary, dialysis.
- In some cases, **corticosteroids** are useful in reducing
  mation.

3:
erative
ixed
phritis.

## NEPHROTIC SYNDROME

Defined as **proteinuria (≥ 3.5 g/day), generalized ede**
**emia,** and **hyperlipidemia.** Approximately one-third of a




**uretics: Site of action.** (Adapted with permission from Katzung BG, Trevor AJ. Basic and Clinical Pharmac

**ses of Nephritic Syndrome**

| description | history/pe | labs/histology |
|---|---|---|
| | **immune complex** | |
| Classically associated with recent group A β-hemolytic **streptococcal infection,** but can be seen | Oliguria, edema, hypertension, tea- or cola-colored urine. | **Low serum C3** that normalizes 6–8 weeks after presentation; **↑ ASO and anti-DNase** |

**pauci-immune (no Ig deposits on immunofluorescence)**

| | | | |
|---|---|---|---|
| omatous<br>n tion of the<br>ory tract **(with haryngeal ement)** and kidney crotizing vasculitis. | Respiratory and sinus symptoms. Cavitary pulmonary lesions bleed and lead to **hemoptysis.** | Presence of **c-ANCA.** Segmental necrotizing glomerulonephritis. | Hig<br>cyt<br>ritu<br>hav |
| essel vasculitis<br>to GPA. **No omas.** | Similar to GPA but **no nasopharyngeal** involvement. | **p-ANCA.** Necrotizing glomerulonephritis. | Glu<br>cyc<br>ritu |
| essel vasculitis. | **Asthma,** sinusitis, skin nodules/purpura, peripheral neuropathy. | **p-ANCA.** ↑ IgE. | Glu |

**anti-gbM disease**

| | | | |
|---|---|---|---|
| progressing<br>ulonephritis with<br>ary hemorrhage;<br>cidence is in men<br>mid-20s. | **Hemoptysis,** dyspnea, possible respiratory failure. **No** upper respiratory tract involvement. | **Linear anti-GBM deposits** on immunofluo escence; iron deficien y anemia; hemosiderin-filled macrophages in sputum; pulmonary infilt ates on CXR. | **Pla**<br>**the**<br>Ma |
| ary<br>ulonephritis;<br>s in boys 5–20 | Asymptomatic hematuria associated with **sensorineural deafness** | GBM splitting on EM. | Pro<br>Ant<br>rec |

B

**indings in nephritic syndromes. (A)** "Lumpy-bumpy" immunofluo-
fectious glomerulonephritis. **(B)** Linear immunofluorescence seen in
(Part A reproduced with permission from Oda T, Yoshizawa N, Yamakami K, et al. The role of
eptor (NAPLR) in glomerulonephritis associated with streptococcal infection, Biomed Biotech-
roduced with permission from Kasper D et al. Harrison's Principles of Internal Medicine, 19th
015.)

**ein and salt restriction,** judicious diuretic therapy, and
ics.
sant medications may be useful for certain etiologies.
nuria and diminish the progression of renal disease in
al scarring (especially in diabetics).
23-polyvalent pneumococcus vaccine (PPV23), as patients
reptococcus pneumoniae infection.

## thiasis

es are most commonly calcium oxalate, but many other
le 2.16-6 and Figure 2.16-12). Risk factors include a ⊕
**fluid intake,** gout, medications (allopurinol, chemother-
, postcolectomy/postileostomy, specific enzyme deficien-
e to alkaline urinary pH and associated hypocitruria), and
. Most common in older men.

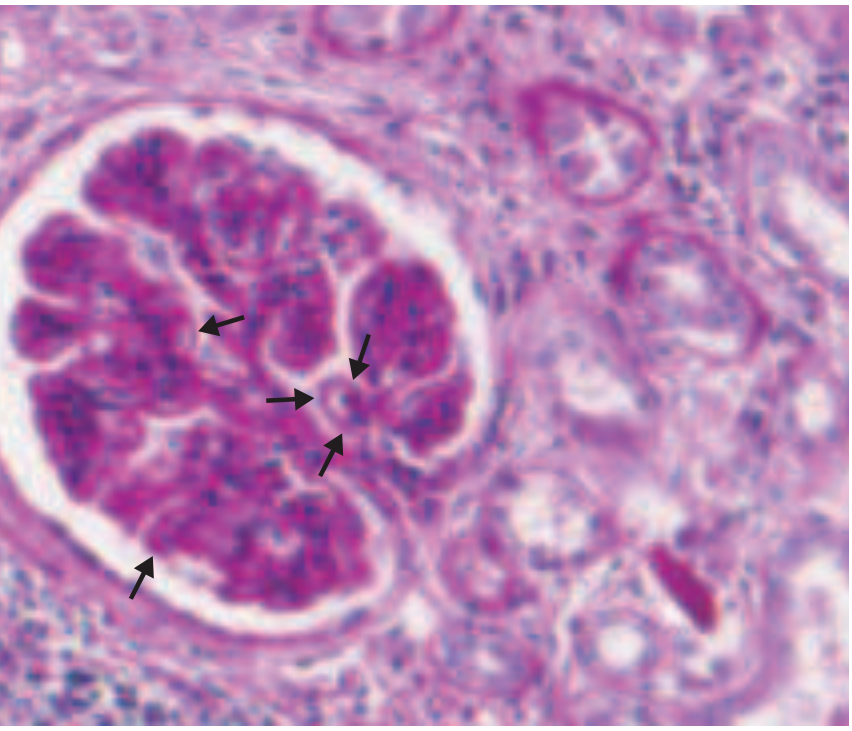
Which b
"stagho
organis

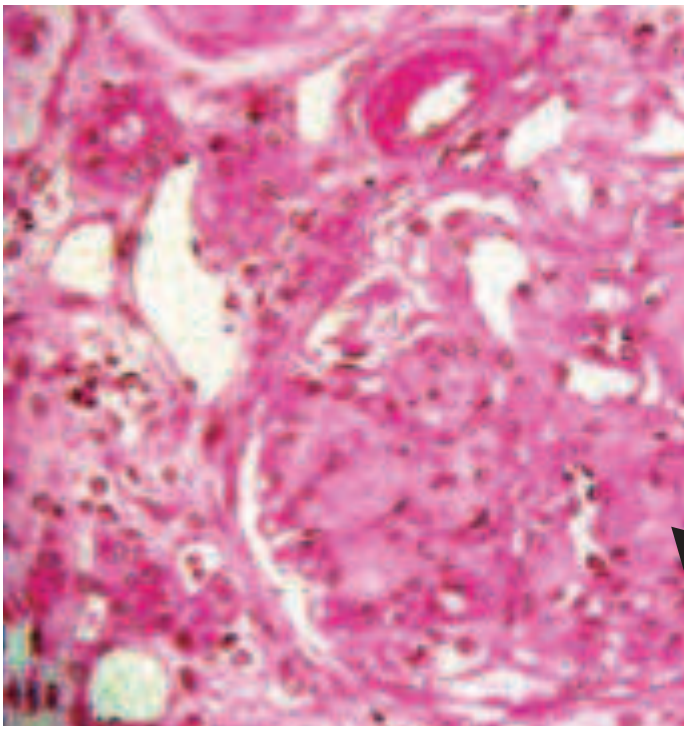
**ute onset of severe, colicky flank pain** that may **radiate to**
**va** and is associated with nausea and vomiting.
ble to get comfortable and shift position frequently (as
with peritonitis, who lie still)

| | | | |
|---|---|---|---|
| **kin disease**). | | | |
| ic, **IV drug use , HIV,** obesity. The mmon cause of ic syndromes in verall. | The typical patient is a young **African American** man with uncontrolled hypertension. | Microscopic hematuria; biopsy shows sclerosis in capillary tufts. | Pre the ↓ p |
| s for ~ 30% of ic syndromes in | Associated with **solid-tumor malignancies,** infections (HBV, malaria), autoimmune (SLE), drugs (NSAIDs, gold). | **"Spike-and-dome"** appearance due to granular deposits of IgG and C3 at the basement membrane. | Pre the |
| aracteristic forms: yalinization and glomerulosclerosis **lstiel-Wilson** . | Patients generally have long-standing, poorly controlled DM with evidence of retinopathy or neuropathy. | Thickened GBM; ↑ **mesangial matrix.** | Tig sug |
| d as WHO types h nephrotic and c. The severity disease often nes overall is. | Proteinuria or RBCs on UA may be found during evaluation of SLE patients. | Mesangial proliferation; subendothelial and/or subepithelial immune complex deposition. | Pre the pro |
| na cell dyscrasia) nfectious or tory) are the mmon. | Patients may have multiple myeloma or a chronic inflamm tory disease (eg, **rheumatoid arthritis,** TB). | Nodular glomerulosclerosis; EM reveals amyloid fib ils; **apple-green** birefringence with Congo red stain. | Pre me tra be mye |

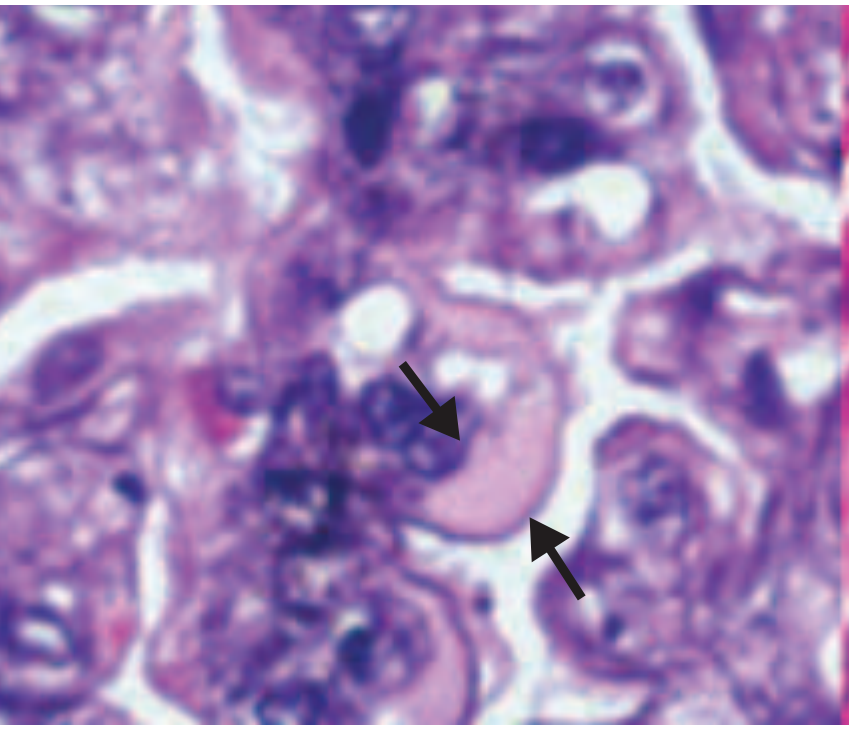


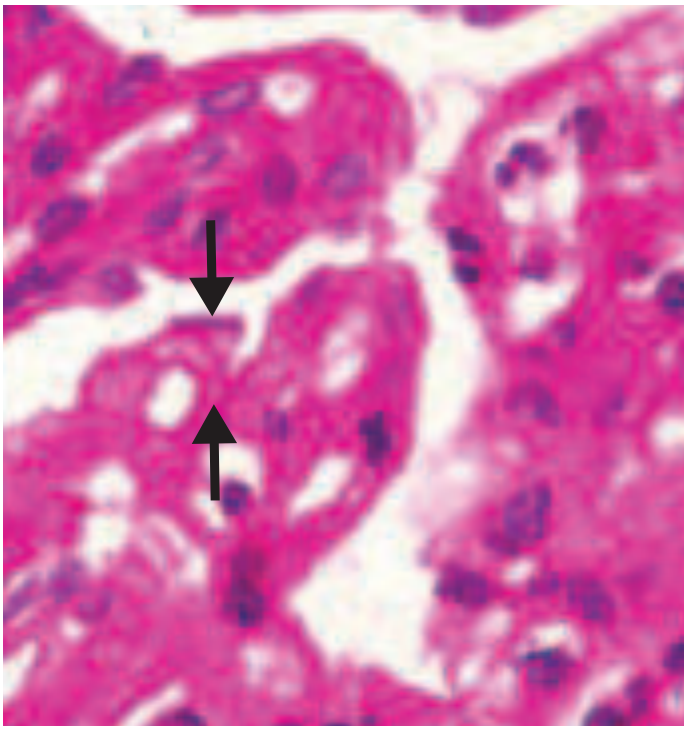
B





D





F

**indings in nephrotic syndromes. (A)** Minimal change disease findings on electron
rrows). **(B)** Focal segmental glomerulosclerosis (arrows). **(C)** Membranous nephropa
Kimmelstiel-Wilson lesions in diabetic nephropathy. **(E, F)** "Tram-track" appearanc
membranoproliferative nephropathy. (Part A reproduced with permission from Le Tet al. First Aid
ed with permission from Nephron. Parts C, E, F reproduced with permission from USMLE-Rx.com. Part D used w

| | | |
|---|---|---|
| Associated with urease-producing organisms (eg, Proteus). Form **staghorn calculi.** Alkaline urine. Radiopaque. | ↑ pH | Hydration surgical re stone. |
| Associated with gout, xanthine oxidase deficien y, and high purine turnover states (eg, chemotherapy). Acidic urine (pH < 5.5). **Radiolucent** on plain film, but detectable with CT. | ↓ pH | Hydration **citrate,** w $HCO_3^-$ in t restriction |
| Due to a defect in renal transport of certain amino acids (**COLA**—cystine, ornithine, lysine, and arginine). **Hexagonal crystals.** ⊕ **urinary cyanide nitroprusside test.** Radiopaque. | ↓ pH | Hydration restriction penicillam |

- Can also consider medical treatment with α-blockers blockers to facilitate stone passage.
- Dietary changes to prevent calcium stones include ↑ important), ↑ **calcium intake,** ↓ dietary protein/oxalate i intake.





th an ↑ risk of cerebral aneurysm, especially in patients
ily history.
**ssive (ARPKD):** Less common but more severe. Presents
oung children with renal failure, liver fibrosis, and portal
n lead to death in the first few years of life.

**turia** are the most common presenting symptoms. Sharp,
ay result from cyst rupture, infection, or passage of renal

ngs include **hypertension, hepatic cysts, cerebral berry**
rticulosis, and mitral valve prolapse.
e large, palpable kidneys on abdominal exam.

graphy (most common) or CT scan (see Figure 2.16-14).
ysts will be present throughout the renal parenchyma, and
will be visualized. Genetic testing by DNA linkage analysis
DPKD2 is available.

**cations and ↓ the rate of progression to ESRD.** Early
UTIs is critical to prevent renal cyst infection. BP control
s necessary to ↓ hypertension-induced renal damage.
al transplantation are used to manage patients with ESRD.







**FIGURE**
CT scan
tion (arr
with nep
sion from T
cine: ACom
NY: McGraw

If a patie
develop
headach
subarach
ruptured

May be asymptomatic, or may present with flank/back pa
abdominal pain, and UTIs.

### Diagnosis

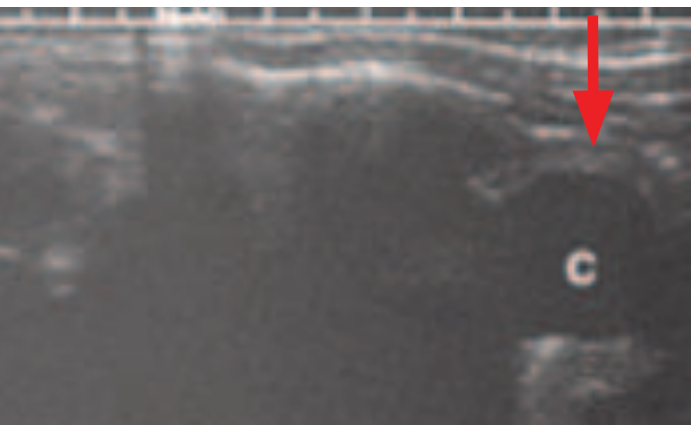
- **Ultrasonography** or CT scan to detect dilation of the ureter (see Figure 2.16-15).
- ↑ BUN and creatinine provide evidence of 2° renal fai

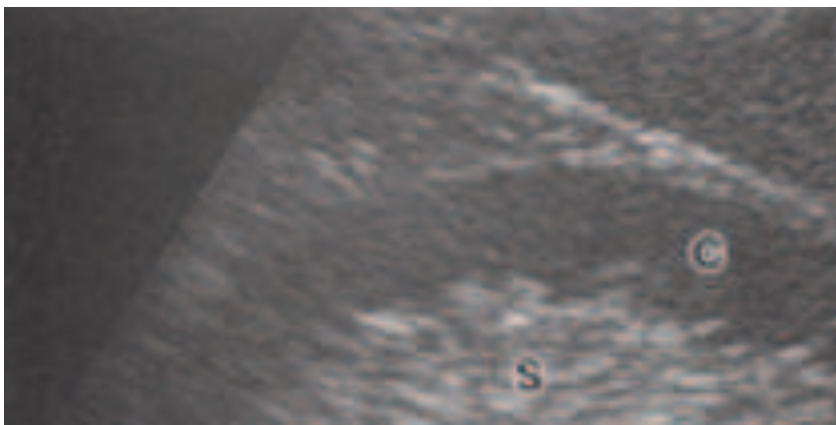
### Treatment

- Surgically correct any anatomic obstruction; use laser tripsy if calculi are causing obstruction.
- Ureteral stent placement across the obstructed area and/or percutaneous nephrostomy tube placement may be appropriate if the urinary outflow tract is not of obstruction. Foley or suprapubic catheters may be urinary tract obstruction (eg, **BPH**).

## Scrotal Swelling

- **Painless** etiologies → hydrocele (remnant of processu cele (dilatation of pampiniform plexus).
- **Painful** etiologies → epididymitis (STDs, prostatitis (twisting of the spermatic cord).



e needed. Will typically resolve. Does have an ↑ risk of hernia.

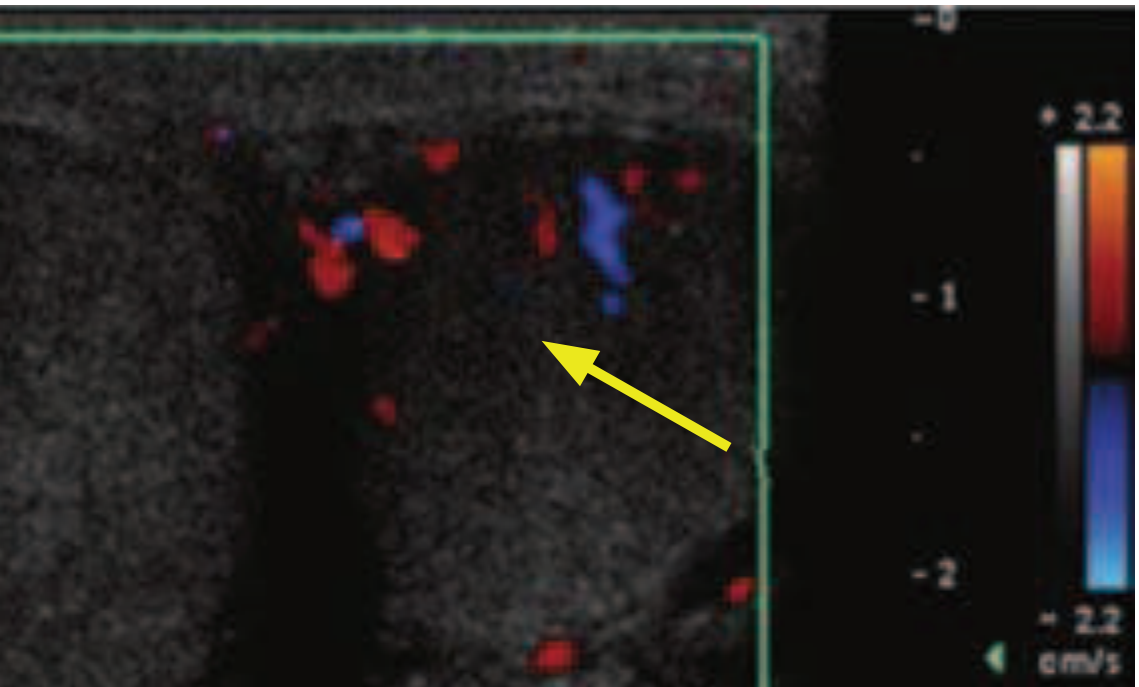
**need surgery if large or symptomatic.**

ntibiotics (tetracycline, fluoroquinolones); NSAIDs; scro-in.

**on:** Immediate surgery (under 6 hours) to salvage testis. detorsion only if surgery is unavailable or if it will not rchiopexy of **both** testes to prevent future torsion.

## Dysfunction

of middle-aged and elderly men. Classified as failure to logical, endocrinologic, neurologic), failure to fill (eg, ure to store (eg, veno-occlusive dysfunction). Risk factors **rosclerosis, medications** (eg, β-**blockers,** SSRIs, TCAs, sion, heart disease, surgery or radiation for prostate cancer, ry.




Q

A 26-y
of dep
altered
and vo
of 7.4,
What
what i

Q

A 17-y
asthm
severe

- **Clinical diagnosis.**
- **Other workup: Testosterone** and **gonadotropin** level Elevated **prolactin** can result in ↓ androgen activity.

### Treatment

- Patients with psychological ED may benefit from psyc discussion and exercises with the partner.
- Oral **sildenafil (Viagra), vardenafil (Levitra), and ta** phosphodiesterase-5 (PDE5) inhibitors that result in cGMP-mediated smooth muscle relaxation and ↑ blo pora cavernosa.
- **Testosterone** is a useful therapy for patients with hypo lar or pituitary origin; it is discouraged for patients w rone levels.
- Vacuum pumps, intracavernosal prostaglandin injec implantation of semirigid or inflatable penile prosth for patients for whom PDE5 therapy fails.

## Benign Prostatic Hyperplasia

Enlargement of the prostate that is a normal part of the is seen in > **80% of men by age 80.** Most commonly pr **years of age.** BPH can coexist with prostate cancer, but I prostate cancer.

### History/Pe

- **Obstructive symptoms:** Hesitancy, weak stream, i incomplete emptying, urinary retention, bladder fullne
- **Irritative symptoms:** Nocturia, daytime frequency, opening hematuria.

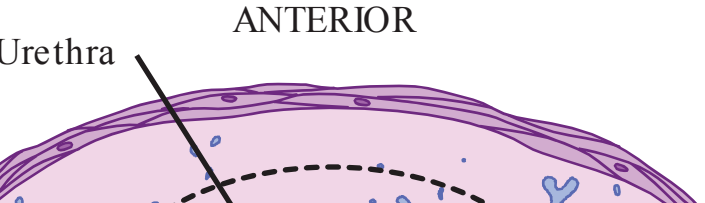
**cancer in men** and the **second leading cause of cancer** lung cancer). Risk factors include advanced age and a ⊕

**omatic,** but may present with **obstructive urinary symp-**
with lymphedema due to obstructing metastases, constitu-
and **back pain due to bone metastases.**
a **palpable nodule** or an area of induration (see Figure
arcinoma is usually not detectable on examination.
suggests prostatitis.

nical findings and/or a markedly ↑ **PSA** (> 4 ng/mL).
osis is made with **ultrasound-guided transrectal** biopsy.
ded by the **Gleason histologic system,** which sums the
o 5) of the two most dysplastic samples (10 is the highest

ses with CT of the abdomen/pelvis and **bone scan** (meta-
w an **osteoblastic** or ↑ bone density).

**g** may be the best approach for elderly patients with low-
many cases of prostate cancer are slow to progress.
**ectomy** is associated with an ↑ risk of incontinence and/




Leading
1. Lu
2. Pro
3. Co
4. Par

A 68-y
of hep
RUQ a
identi

with prostate cancer.

## Bladder Cancer

The second most common urologic cancer and the most
tumor of the urinary tract; usually a **transitional cell carci**
18**).** Most prevalent in men during the sixth and seventh d
include **smoking,** diets rich in meat and fat, schistosom
ment with cyclophosphamide, and occupational exposure

### History/Pe

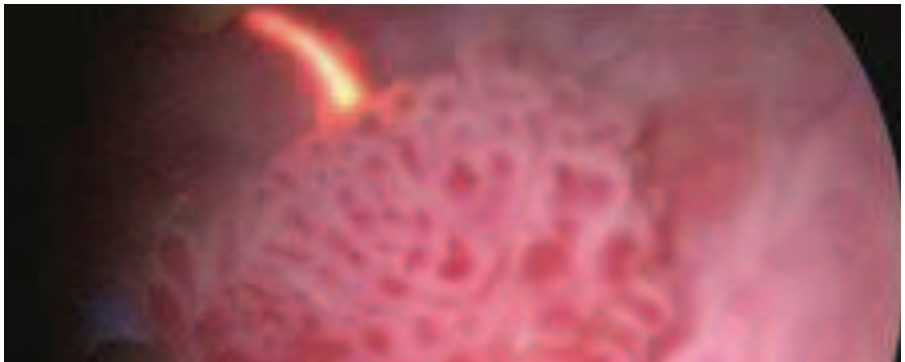
- **Gross hematuria** is the most common presenting symp
- Other urinary symptoms, such as frequency, urgenc
  also be seen, but most patients are asymptomatic in th
  ease.

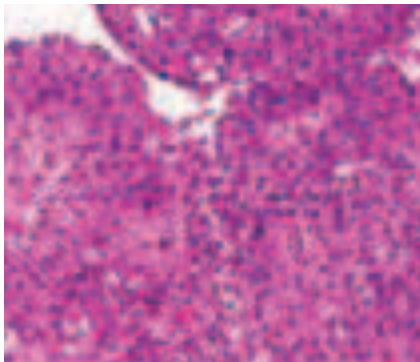
### Diagnosis

- **Cystoscopy with biopsy is diagnostic** and is recomme
  tion of older adults to rule out malignancy.
- **UA** often shows hematuria (macro- or microscopic).
- **Cytology** may show dysplastic cells.
- **MRI, CT,** and bone scan are important tools with w
  sion and metastases.

A

st-
d
ation



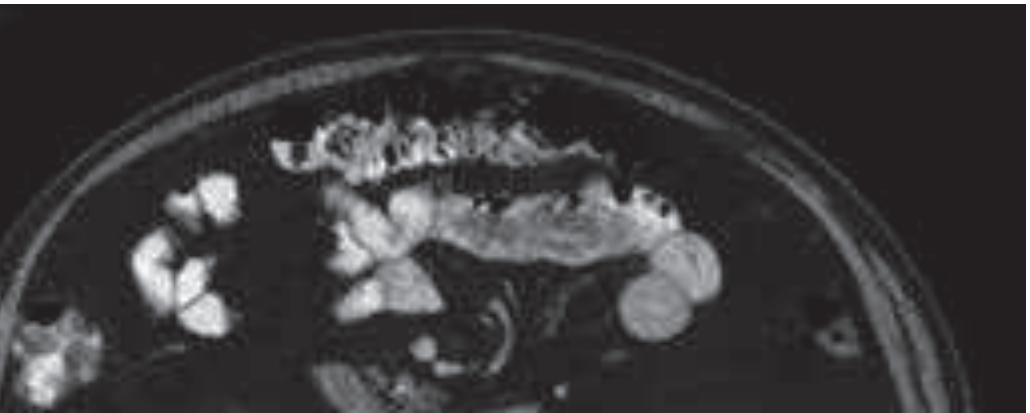


# l Carcinoma

from tubular epithelial cells (~ 80–90% of all malignant ey). Tumors can spread along the renal vein to the IVC e to lung and bone. Risk factors include **male gender, cquired cystic kidney disease in ESRD,** and von Hippel–

include **hematuria, flank pain,** and a **palpable flank** disease can present with weight loss and malaise.
ave **fever** or other constitutional symptoms. Left-sided var- seen in men (due to tumor blockage of the left gonadal ties into the left renal vein; the right gonadal vein empties IVC).
**mon at presentation,** but **polycythemia** due to ↑ erythro- on can be seen in 5–10% of patients.

(see Figure 2.16-19) to characterize the renal mass and odes/metastases. Ultrasonography rarely used. Diagnosis istology on nephrectomy specimen.



The clas
is hema
flank ma
with all

In a mid
left-side
carcinor

A heterogeneous group of neoplasms. Some 95% of testi
from **germ cells,** and **virtually all are malignant. Crypt**
ated with an ↑ risk of neoplasia in both testes. **Klinefelte**
risk factor. Testicular cancer is the most common malign
years of age.

### History/Pe

- Patients most often present with **painless enlargement**
- Most testicular cancers occur between ages 15 and
  have a peak incidence between 40 and 50 years of age.

### Diagnosis

- **Testicular** ultrasonography.
- **CXR** and abdominal/pelvic CT to evaluate for metasta
- **Tumor markers** are useful for diagnosis and in m
  response. See Table 2.16-7.

### Treatment

- Radical orchiectomy.
- Seminomas are **exquisitely radiosensitive** and also respo
- Platinum-based chemotherapy is used for nonsemin
  tumors.

**TABLE 2.16-7. Tumor Markers in Testicular Cancer**

| Type | Tumor |
|---|---|
| **Germ cell tumors (95% of all testicular tu** | **Mo** |
| Seminoma (most common testicular tumor) | ↑ placental ALP |

494 Burns

494 Postoperative Fever

494 Shock

497 Thermal Dysregulation

497 Hypothermia

497 Hyperthermia

498 Bites and Stings

498 Toxicology

498 Carbon Monoxide Poisoning

499 Common drug Interactions/Reactions

501 drug overdose

502 Major drug Side Effects

502 Management of drug Withdrawal

502 Recognition of drug Intoxication

504 Vitamin Deficiencies

n Trauma

asics

Figure 2.17-1 illustrates the 1° issues to be surveyed on patient.

## 2° SURVEY

- After the patient's ABCDEs are managed, conduct a fu ination.
- For hemodynamically unstable patients, or those wit trauma to the abdomen or chest, do a focused abdom trauma **(FAST)** scan (Figure 2.17-3). Hemoperitoneu ate surgical consultation for operative management; warrants immediate pericardiocentesis to prevent card Figure 2.17-4).
- After urethral injury has been ruled out, place a Fole tor urine output in hemodynamically unstable patient those undergoing surgery.
- Order radiology studies on the basis of the patient asses
  - CXR for thoracic trauma.
  - Head CT (clinical decision guidelines assess factors mental status, suspected skull fracture, and mechan
  - C-spine CT is needed for all patients with neck neurologic findings, or depressed mental status.
- Pertinent labs should address mechanism of injury, i dose, and medical history.

**TABLE 2.17-1. Glasgow Coma Scale Scoring**

| Score | Eye Opening Response (4) | Verbal Response (5) |
|---|---|---|
| 6 | | |
| 5 | | Oriented |

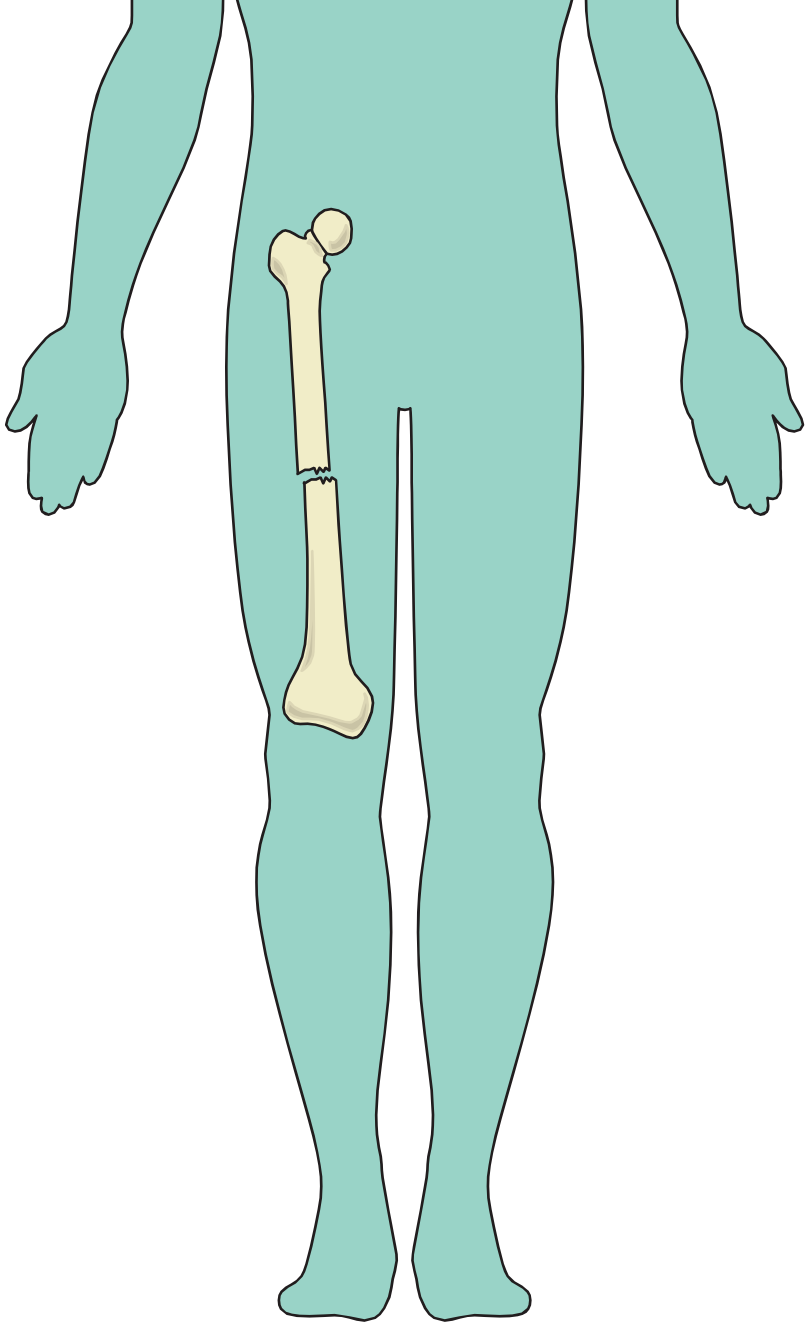
trauma.
nothorax suspected, place

. Splint long-bone

od to unstable patients.

motor/sensory function in

pect for additional injuries.
nary survey to avoid

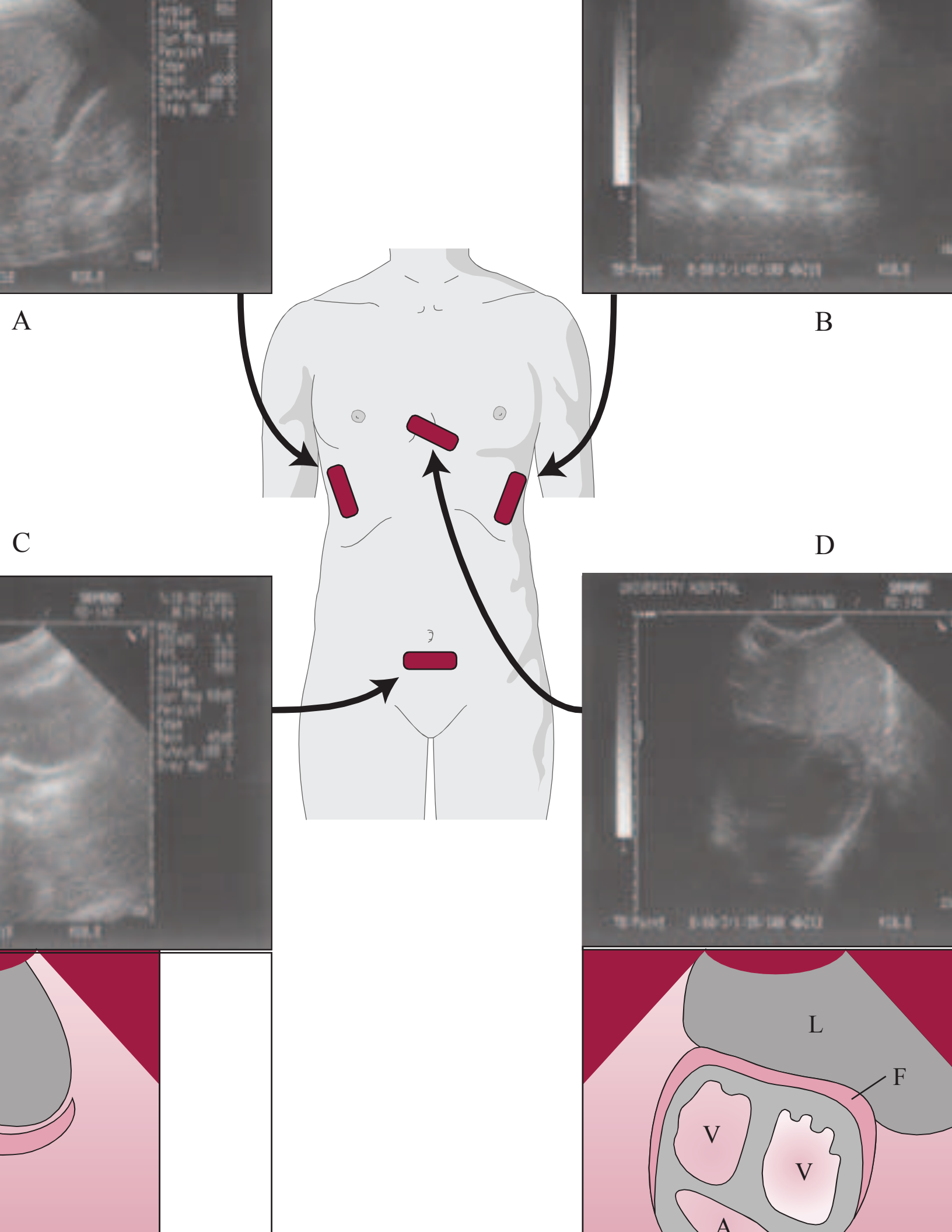


Arough
can be r
pulses. C
an SBP o
of 70 mr
80 mm

Suspect
of blood
perineal
scrotum
prostate
retrogra
urethral

**imary trauma survey.**




A
B
C
D
L
F
V
V
A



**rdiac tamponade.** Echocardiogram in a patient with cardiac tam-
ricardial effusion (PE) with right atrial (arrow) and right ventricular
produced with permission from Hall JB et al. Principles of Critical Care, 3rd ed. New York, NY:

## ng Trauma

tment of penetrating trauma depend on the location and

violate the platysma are considered true penetrating neck

patients early before the airway becomes more compro-

cal exploration of neck wounds is indicated if shock and
ge are present.
s may include angiography of the aorta and carotid/cere-
scan of the neck with or without CT angiography, Dop-
phy, contrast esophagogram, esophagoscopy, or bronchos-
should be guided by mechanism of injury and patient

ts with penetrating thoracic injuries require immediate
wed by assessment and treatment of the life-threatening
d above. Empiric placement of bilateral chest tubes may

ful in settings of equivocal results or in resource-poor s

### MUSCULOSKELETAL

- Neurovascular assessment is critical; check pulses, n sensory function.
- Arteriography and surgical management are required lar injuries.
- Nerve injuries generally require surgical repair.
- **Early wound irrigation** and **tissue debridement,** no are the most important steps in the treatment of co However, do administer antibiotics and **tetanus proph**

## Blunt and Deceleration Trauma

### HEAD

- Look for signs of ↑ ICP such as bradycardia, hype depression, fixed and dilated pupil(s), vomiting, and/o ↑ ICP with head elevation, hyperventilation, and IV surgical decompression.
- A rapid-deceleration head injury causes **coup-cont** which bleeding is noted both at the site of impact a point of impact.
- Diffuse axonal injury often occurs with rapid-decele CT characteristically shows **blurring and punctate h the gray–white matter junction.** Reduce 2° injury edema and increases in ICP.
- **Epidural hematomas:** Lenticular or biconvex in shap Figure 2.17-5A). The hemorrhage is often from the

Ehlers-
to a

B

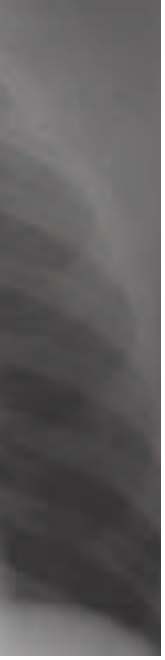
**ute epidural and acute subdural hematoma. (A)** Noncontrast CT
l acute epidural hematoma. Note the characteristic **biconvex** shape.
nonstrating a right acute holohemispheric subdural hematoma. Note
**ntic** shape. (Part A reproduced with permission from Doherty GM. Current Diagnosis &
York, NY: McGraw-Hill; 2010. Part B reproduced with permission from Chen MY et al. Basic
McGraw-Hill; 2004.)

al disruption is most often caused by deceleration shear-
cal findings include respiratory distress, hemoptysis, ster-
and subcutaneous emphysema. Radiographs may show a
orax or pneumomediastinum (see Figure 2.17-6).
njury (aka myocardial contusion) may present as a new
block, ectopy or dysrhythmia, or hypotension. Serum car-
are often elevated. Intervention is rarely required.
usion may lead to hypoxia due to damage to capillaries
tra- and extravascular fluid. Hypoxia tends to worsen with
Look for patchy alveolar opacities on CXR. Maintain ade-
and control pain. More common in children because of
ctive chest wall.



Q

A 25-y
ED ho
agains
by his
and hi
exami
wound



uption.
man
nicle
medias-
a to the
ratra-
) blur-
ion of
teration
Repro-
phries RL.
edicine, 7th

A

ration
auma
le
al
er

- **CXR** reveals a **widened mediastinum** (> 8 cm), **loss o**
  **ral cap,** deviation of the trachea and esophagus, and d
  main stem bronchus (Figure 2.17-7).
- Ultrasonography can help with diagnosis of concurre
  ponade.
- If patient is stable, CT should be obtained to confi
  findings. Can be done if patient is unstable for CT.

### Treatment

Basic trauma management (ABCs); emergent surgery for a

## Flail Chest

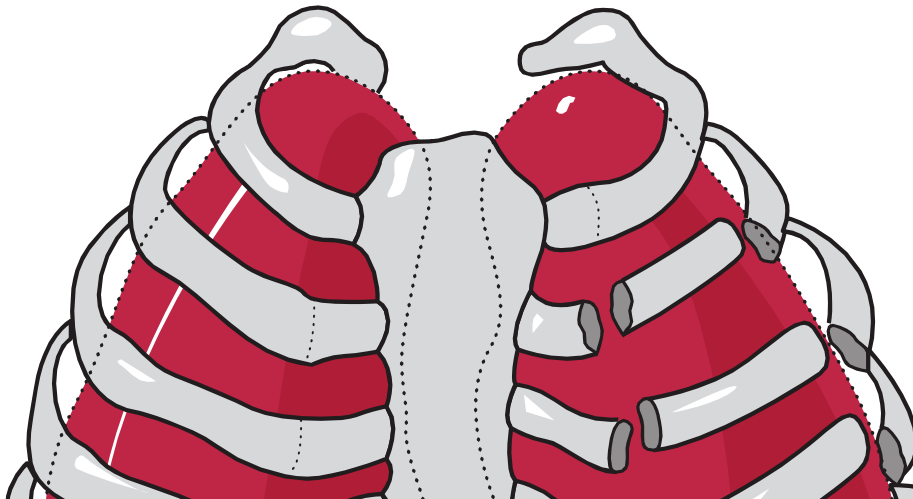
**Three or more adjacent ribs fractured at two points,**
inward movement of the segment with inspiration (Figure

### History/Pe

Presents with crepitus and abnormal chest wall moveme
wall movement may not be appreciated if the patient is
pain.

### Diagnosis

Primarily clinical, although CXR and $O_2$ saturation are he



the **most commonly injured organ following blunt ma,** with **liver** and **hollow viscus** injuries following in r. Symptoms are consistent with signs of blood loss and sion, tachycardia, and peritonitis. Suspect spleen or liver er rib fractures are present.

is susceptible to compression injury as a result of its posi- the spine. Look for retroperitoneal air on AXR, and con- sis with CT, which can also rule out a duodenal hema-

y or bowel hematoma should be suspected after a direct handlebar injury).

**rupture** may occur with blunt or penetrating trauma. be present; radiographs may demonstrate an elevated or abdominal viscera in the thorax.

the **most commonly injured GU organ in trauma,** with g renal contusion, laceration, fracture, and pedicle injury. cally stable patients, abdominal blunt trauma can be diag- T scan, CT scan, and serial abdominal exams.

cally unstable patients, abdominal blunt trauma should be nediate exploratory laparotomy to look for organ injury or bleeding.

cur after high-speed traumas such as motor vehicle acci- heights. Require prompt stabilization with an external evaluation by a trauma or orthopedic surgeon in light of -threatening hemorrhage.

n an unstable pelvis upon manipulation.

**Kehr sig**
may lea
shoulde
diaphrag
phrenic
the brac

Unstabl
trauma
explorat

Look for
in restra
possible
to decel
(Figure 2



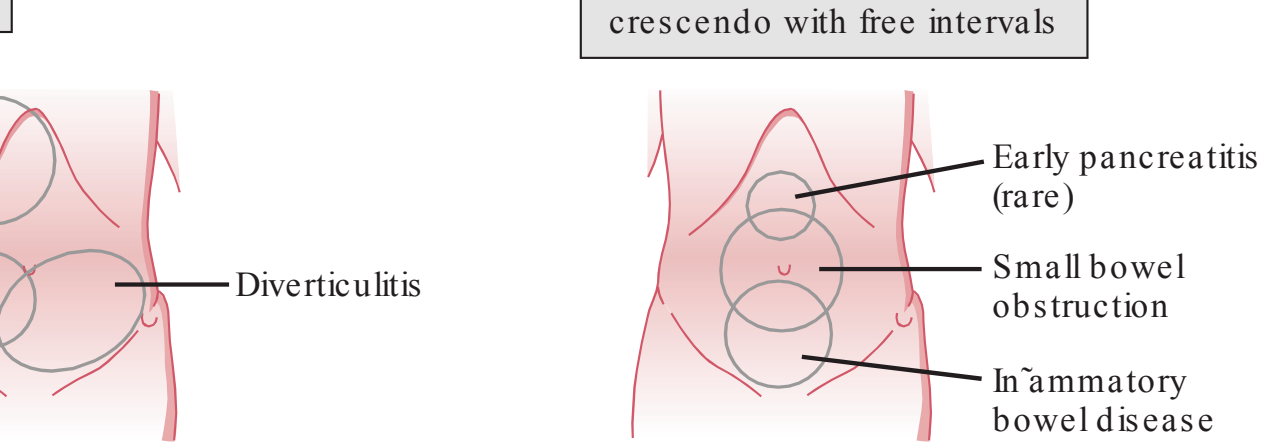
Table 2.17-2 summarizes the basic management of cardia
acute setting.

## Acute Abdomen

Acute-onset abdominal pain has many potential etiologi
immediate medical or surgical intervention. Sharp, focal p
and guarding generally implies a parietal (peritoneal) eti
achy, and midline or diffuse pain is commonly of visc
Patients with parietal pain generally prefer immobility,
visceral pain are often unable to lie still. Figure 2.17-10
causes of acute abdomen.

**t of Cardiac Arrhythmias**[a,b]

| | t reat Ment |
|---|---|
| | nitiate CPR. Give epinephrine or vasopressin; simultaneously search for the underlying c<br>5 **T's** mnemonics) and provide empiric treatment. |
| | nitiate CPR. Defib illate with 120–200 J (biphasic) immediately → defib illate again → ep<br>→ defibrillate → amiodarone → defib illate → epinephrine. |
| | f unstable, perform synchronized electrical cardioversion.<br>f stable, control rate with vagal maneuvers (Valsalva maneuver, carotid sinus massage, c<br>f resistant to maneuvers, give adenosine followed by other AV-nodal blocking agents (C<br>rhythm fails to convert. |




**Acute abdomen.** The location and character of pain are helpful in the the acute abdomen. (Reproduced with permission from Doherty GM et al. Current Diag-ed. New York, NY: McGraw-Hill; 2010.)

tion to the historical features in the acute abdominal pre-

ynecologic history in women (including last menstrual cy, and any STD symptoms). If a patient has abdominal al motion tenderness, there should be a low threshold to **nflammatory disease (PID).**

ds to sudden onset of diffuse, severe pain, usually with ty.

ds to acute onset of severe, radiating, colicky pain. Patients f obstipation (failure to pass stool or gas) or bilious emesis. bowel obstruction in patients with a history of abdominal

eads to gradual onset (over 10–12 hours) of constant, ill-

**toms** include the following:

usea, vomiting, changes in bowel habits, hematochezia, uggest GI etiologies.

ugh suggest pneumonia.

yuria, and costovertebral angle tenderness point to a GU

Pneumo
quadran
diaphrag

Q

A 22-y
the ED
in whi
She re
and ha
heart
has ↓
neck v
on the
likely

Q

A 36-y
ED fol
in whi
passer
fractur
develo
and a
purple
his arm
most

Tr ea Tmen T

- **Hemodynamically unstable patients must have eme agement.**
- In stable patients, expectant management may inclu tube placement (for decompression of bowel in the se or acute pancreatitis), IV fluids, placement of a Foley urine output and fluid status), and vital sign monitorin inal examinations and serial labs.
- Broad-spectrum antibiotics for patients with perforatic May be indicated for patients with infectious processe tis, diverticulitis, and pyelonephritis.
- Type and cross all unstable patients and those in whon tial hemorrhage.

## Acute Appendicitis

The inciting event is obstruction of the appendiceal lum inflammation and infection. Rising intraluminal pressu compromise of the appendix, ischemia, necrosis, and p Etiologies include hypertrophied lymphoid tissue (55–65 foreign body, tumor (eg, carcinoid tumor), and parasites. the early teens (most patients between 10 and 30 years of to-female ratio is 2:1.



atypical presentations that may result in misdiagnosis and

nical impression.
mild leukocytosis (11,000–15,000 cells/μL) with left shift,
w RBCs and/or WBCs.
agnosis is unequivocal, no imaging studies are necessary.
es include the following:
**h PO and IV contrast (95–98% sensitive):** Periappendi-
g or fluid; enlarged appendix (see Figure 2.17-12).
**phy:** An enlarged, noncompressible appendix. Preferred
nd pregnant women.

ld be NPO and receive IV hydration, analgesia, antiemet-
cs with anaerobic and gram-⊖ coverage.
or laparoscopic appendectomy is the definitive treatment.
not found, complete exploration of the abdomen is per-

minister antibiotics until the patient is afebrile with a nor-
ount; the wound should be closed by delayed 1° closure.
with broad-spectrum antibiotics and percutaneous drain-
appendectomy should be performed 6–8 weeks later.



- **"Ha**
  wan
  othe
- **Pso**
  the
- **Obt**
  rota
  RLQ
- **Rov**
  the

involved. The area is painful, and blisters are present.
- **Third degree:** The epidermis, the full thickness of the tially deeper tissues are involved. The area is painless,

### History/Pe

- Patients may present with obvious skin wounds, b destruction may not be visible, especially with electrica
- Conduct a thorough airway and lung examination to injury.
- Maintain a high index of suspicion for carbon mon patients with inhalation injury or involvement in a clos
- Consider cyanide poisoning in closed-space fires with textiles.

### Diagnosis

- Assess ABCs. If evidence of thermal or inhalation inju way, intubate.
- Be vigilant for shock, inhalation injury, and carbon m Obtain a CXR and a carboxyhemoglobin level.
- Evaluate the percentage of BSA (% BSA) involved (see

### Treatment

- Supportive measures; tetanus, stress ulcer prophylax analgesia.
- For second- and third-degree burns, fluid repletion **formula** is critical; adjust repletion on the basis of a losses to maintain at least 1 cc/kg/hr of urine output.



artial-thickness burn over critical areas (face, hands, feet, neum, major joints).
tial burns; chemical, electrical, or lightning injury; inha-

sychosocial or rehabilitative care needs.

## ative Fever

of all postoperative patients. Remember the mnemonic **W**ounds, **W**alking, and **W**onder drugs." Another **"W"** of er is "**W**omb" in OB/GYN (Table 2.17-3).
stoperative fever with incentive spirometry, pre- and post-otics when indicated, short-term Foley catheter use, early d DVT prophylaxis (eg, anticoagulation, compression

stoperative day 3 are unlikely to be infectious unless Clos-nolytic streptococci are involved.

**uate tissue-level oxygenation to maintain vital organ** tiple etiologies are differentiated by their cardiovascular t options (see Table 2.17-4).

**toperative Fever Timing**

| **t iMe Fr a Me** | **e t io l o g y** | **Mn e Mo n ic** |
|---|---|---|

Heart ra
change
only afte
volume.

Avoid p
until ad
been pr

Maligna
maligna
be ruled
hyperth
would b
and NM
conditio

Followin
animals

| | | | | |
|---|---|---|---|---|
| disease (severe mitral regurgitation, VSD), MI (> 40% of left ventricular function). | | | | possible. Give with pressors s hypotensive) c hypotensive). |
| Cardiac tamponade, tension pneumothorax, massive pulmonary embolism. | ↓ | ↑ | ↑ | Treat the unde Pericardiocent pneumothora |
| | ↑ | ↓ | ↓ | |
| Bacteremia, especially gram-⊖ organisms. | | | | Administer bro Measure centra and give fluid u (norepinephrin needed. Obtai administration |
| Bee sting, medications, food allergy. | | | | 1:1000 epinep adjuncts $H_1/H_2$ steroids. |
| Pancreatitis, burns, trauma. | | | | Manage under |
| Brain or spinal cord injury. | | | | Maintain press pressor suppo |

mmon at < 30°C (< 86°F). The classic sign is the **J wave**
e). Also monitor electrolytes and acid-base balance.
scitation efforts until the patient has been warmed.

> 40°C (> 104°F).

gnant hyperthermia, NMS, poisoning, overdose, with-
, environmental.
s, meningitis/encephalitis, tetanus, typhoid, malaria.
oid storm, pheochromocytoma, diabetic ketoacidosis.
othalamic stroke, seizures, CVA.

tient with cold water, wet blankets, and ice. Give a benzo-
t shivering. Rule out causes of fever such as infection or

## Stings

es the management of common bites and stings. Table
he recommended prophylaxis for rabies and tetanus.

## gy

it does n
arterial P

Bites inv
resulting
be sutur
and mo
infection

Conside
tooth fra

Commo
Pasteure
canimor
staphylo
species.

| | |
|---|---|
| se. | scorpion stings); IV scorpion-specific a tibody. |
| soning. | Antivenom is the mainstay of treatment. Keep below the heart. Compression bands help, but contraindicated. Suction and incision are dang be done. |
| abies/tetanus. | Amoxicillin/clavulanate for puncture wounds, high-risk or immunocompromised patients. |
| | Amoxicillin/clavulanate unless very minor. |
| infection; not known to carry rabies. | Local wound care. |

- **Suspect smoke inhalation** in the presence of **singe burns, hoarseness, wheezing,** or **carbonaceous sputu**

eatment
r used
e
f

Diagnosis

- Check an ABG and serum carboxyhemoglobin level nonsmokers and < 10% in smokers).
- Check an ECG in the elderly and in patients with a histo

**TABLE 2.17-6. Rabies and Tetanus Precautions**

| Suspected pathogen | Exposure | Prophyla |
|---|---|---|
| Rabies | Bites from dogs, cats, ferrets, skunks, raccoons, **bats,** | If an animal displays signs of ra of human rabies immune glo rabies vaccine over 14 days i |

nes drug interactions and reactions that are commonly



Activate
cases of
hours) o
to it (no
hydroca

## 

arizes antidotes and treatments for substances commonly
rdoses and intoxications. If a patient is unresponsive, it is
ally treat with a dose of naloxone.

## EFFECTS

es the major side effects of select drugs.

**g Interactions and Reactions**

| eaction | drugs |
|---|---|
| zymes | **Barb**iturates, **S**t. John's wort, **P**henytoin, **R**ifampin, **G**riseofulvin, **Ca**<br>**P**hen-phen and **R**efuses **G**reasy **C**arbs." |
| zymes | Quinidine, cimetidine, ketoconazole, INH, grapefruit, erythromycin, |
| enzymes | Benzodiazepines, amide anesthetics, metoprolol, propranolol, nifed<br>theophylline, warfarin, barbiturates. |
| ty | Quinidine, cimetidine, amiodarone, CCBs. |
| hin-binding sites | Warfarin, ASA, phenytoin. |
| | Ibuprofen, quinidine, methyldopa, chemotherapeutic agents. |

| | |
|---|---|
| | Succimer, dimercaprol. |
| | Glucagon. |
| | Urine alkalinization, dialysis, activated charcoal, supportive care. |
| | Flumazenil (can precipitate withdrawal, mainly used for acute ingestion). |
| | Calcium gluconate, methocarbamol. |
| | 100% $O_2$, hyperbaric $O_2$. |
| | Penicillamine. |
| | Hydroxycobalamin, amyl nitrate, sodium nitrate, sodium thiosulfate. |
| | Normalize $K^+$ but avoid giving $Ca^{2+}$, $Mg^{2+}$, or lidocaine (for torsades); anti-d |
| | Protamine sulfate. |
| | Pyridoxine. |
| | Deferoxamine. |
| | Succimer, CaEDTA, dimercaprol. |
| freeze) | EtOH, fomepizole, dialysis, calcium gluconate for ethylene glycol. |
| | Methylene blue. |
| | Naloxone. |
| | Urine alkalinization, dialysis, activated charcoal. |

| | |
|---|---|
| | discoloration, arrhythmias, hypo-/hyperthyroidism, corneal deposition. |
| | Fever/rigors, nephrotoxicity, bone marrow suppression, anemia. |
| | Sedation, acute dystonic reaction, akathisia, parkinsonism, tardive dyskinesia, **N** |
| e) | Inhibition of P-450 enzymes. |
| | Thrombocytopenia, megaloblastic anemia. |
| | Asthma exacerbation, masking of hypoglycemia, impotence, bradycardia, AVbl |
| | Sedation, dependence, respiratory depression. |
| | GI upset, malabsorption of vitamins and medications. |
| | Induction of P-450 enzymes, **agranulocytosis,** aplastic anemia, liver toxicity. |
| | Peripheral edema, constipation, cardiac depression. |
| | **Gray baby syndrome,** aplastic anemia. |
| | Nephrotoxicity, acoustic nerve damage. |
| | Dry mouth; **severe rebound headache and hypertension.** |
| | Agranulocytosis. |
| | Mania, hyperglycemia (acute), immunosuppression, bone mineral loss, thinning myopathy, cataracts (chronic). |
| | Myelosuppression, **hemorrhagic cystitis.** |

| |
|---|
| Drug-induced SLE. |
| Retinopathy. |
| Peripheral neuropathy **(prevent with pyridoxine/vitamin $B_6$),** hepatotoxicity, inhibition<br>Seizures with overdose, hemolysis in G6PD deficien y. |
| **Hypertensive tyramine reaction, serotonin syndrome** (with meperidine). |
| Blindness, anion-gap metabolic acidosis. |
| Hepatic fib osis, pneumonitis, anemia. |
| ⊕ Coombs test, drug-induced SLE. |
| Disulfi am reaction, vestibular dysfunction, **metallic taste.** |
| **Cutaneous flushin .** |
| Hypotension, tachycardia, headache, tolerance. |
| Drug-induced SLE. |
| Hypersensitivity reactions. |
| Nystagmus, diplopia, ataxia, arrhythmia (in toxic doses), **gingival hyperplasia,** hirsutism |
| First-dose hypotension. |
| Drug-induced SLE. |

| | |
|---|---|
| | Megaloblastic anemia, leukopenia, granulocytopenia. |
| | Teratogenicity leads to neural tube defects; rare fatal hepatotoxicity. |
| | Nephrotoxicity, ototoxicity, **"red man syndrome"** (histamine release; not an alle |
| | Severe myelosuppression. |
| | Peripheral neuropathy, paralytic ileus. |

## DRUG WITHDRAWAL

marizes common drug withdrawal symptoms and treat-

**mptoms and Treatment of Drug Withdrawal**

| Withdrawal Symptoms | t |
|---|---|
| Tremor (6–12 hours).<br>Tachycardia, hypertension, agitation, seizures (within 48 hours).<br>Hallucinations, **DTs**—severe autonomic instability leading to tachycardia, hypertension, delirium, and possibly death (within 2–7 days). Mortality is 15–20%. | **Benzodiazepines** (can re<br>haloperidol for hallucinati<br>multivitamin replacement<br>most alcoholics are deficie |
| Anxiety, seizures, delirium, tremor; tachycardia and hypertension. | **Benzodiazepines.** |

## nctions and Deficiencie

| Signs/Symptoms of deficiency |
|---|
| Night blindness, dry skin. |
| Beriberi (polyneuritis, dilated cardiomyopathy, high-output CHF, edema), Wernicke-Korsa |
| Angular stomatitis, cheilosis, corneal vascularization. |
| Pellagra (diarrhea, dermatitis, dementia). |
| Dermatitis, enteritis, alopecia, adrenal insufficie y. |
| Convulsions, hyperirritability; required during administration of INH. |
| Macrocytic, megaloblastic anemia; neurologic symptoms (eg, optic neuropathy, subacut egeneration, paresthesias); glossitis. |
| Scurvy (eg, swollen gums, bruising, anemia, poor wound healing). |
| Rickets in children (bending bones), osteomalacia in adults (soft bones), hypocalcemic te |
| fragility of RBCs. |
| Neonatal hemorrhage; ↑ PT and aPTT, normal BT. |
| Dermatitis, enteritis. Can be caused by ingestion of **raw eggs** or antibiotic use. |
| The **most common vitamin deficien y in the United States.** Sprue; macrocytic, megalo eurologic symptoms. |

518 Neurology
520 Obstetrics
521 Gynecology
522 Pediatrics
523 Psychiatry
524 Pulmonary
526 Renal/Genitourinary
528 Selected Topics In Emergency M
530

| | |
|---|---|
| | β-blockers, calcium channel blockers (CCBs), d |
| nt that leads to flushing and pru itus. | Niacin. |
| ctive cardiomyopathy. | A systolic ejection murmur heard along the lat<br>↑ with ↓ preload (Valsalva maneuver). |
| | Austin Flint murmur, a diastolic, decrescendo, l<br>murmur that is best heard sitting up; ↑ with ↑<br>maneuver). |
| | A systolic crescendo/decrescendo murmur tha<br>↑ with ↑ preload (squatting maneuver). |
| | A holosystolic murmur that radiates to the axil<br>(handgrip maneuver). |
| | A diastolic, mid- to late, low-pitched murmur p<br>snap. |
| and atrial flut er. | If unstable, cardiovert. If stable or chronic, rate<br>β-blockers. |
| ation. | Immediate cardioversion. |
| | An autoimmune reaction with fever, pericardit<br>2–4 weeks post-MI. |
| osystolic murmur at the left sternal | Treat existing heart failure, and replace the tric |

| | |
|---|---|
| c test? | |
| with stable angina can exercise to 85% of d heart rate. | Exercise stress treadmill with ECG. |
| an with left bundle branch block and severe nstable angina. | Pharmacologic stress test (eg, dobuta |
| ia during stress testing. | Angina, ST-segment changes on ECG, |
| ng MI | ST-segment elevation (depression me T waves, and Q waves. |
| MI. | Anterior wall (LAD/diagonal), inferior oblique, RCA/marginal), septum (LAD |
| angina at rest and ST-segment elevation with es. | Prinzmetal angina. |
| ssociated with silent MIs. | CHF, shock, and altered mental status |
| monary embolism. | Spiral CT with contrast. |
| | Reverses the ef ects of heparin. |
| | The coagulation parameter af ected b |
| a family history of sudden death collapses and | Hypertrophic cardiomyopathy. |
| xis regimens. | Oral surgery—amoxicillin for certain s procedures—not recommended. |
| | Stasis, hypercoagulability, endothelia |

| | |
|---|---|
| of diabetes presents with a red, y. | Cellulitis. |
| | Pemphigus vulgaris. |
| | Bullous pemphigoid. |
| esents with dirty, velvety patches on | Acanthosis nigricans. Check fasting blood gluc diabetes. |
| | Varicella zoster. |
| | Lichen planus. |
| | Erythema multiforme. |
| ric pattern in areas where skin comes welry. | Contact dermatitis. |
| nd many smaller ones in a treelike | Pityriasis rosea. |
| ons on the chest and back; KOH prep ”appearance. | Tinea (pityriasis) versicolor. |
| uggestive of melanoma. | Asymmetry, border irregularity, color variation changing appearance. |
| exposure that can lead to | Actinic keratosis. |

| | |
|---|---|
| use of hypothyroidism. | Hashimoto thyroiditis. |
| no o thyroiditis. | High TSH, low $T_4$, antibodies to thyroi |
| al myxedema, and ↓ TSH. | Graves disease. |
| use of Cushing syndrome. | Iatrogenic corticosteroid administrati<br>cause is Cushing disease. |
| ectomy presents with signs of hypocalcemia | Hypoparathyroidism (iatrogenic). |
| s, psychiatric overtones." | Signs and symptoms of hypercalcemi |
| lemia, and metabolic alkalosis. | 1° hyperaldosteronism (due to Conn s<br>hyperplasia). |
| h tachycardia, wild swings in BP, headache,<br>ental status, and a sense of panic. | Pheochromocytoma. |
| first in t eating pheochromocytoma, α- or | α-antagonists (phenoxybenzamine). |
| y of lithium use presents with copious<br>e. | Nephrogenic diabetes insipidus (DI). |
| DI. | Administration of DDAVP and free-wa |
| nt with significa t pain presents with<br>rmal volume status. | Syndrome of inappropriate antidiuret<br>stress. |
| associated with lactic acidosis. | Metformin. |

| | |
|---|---|
| wing 95% confiden e interval (CI) for<br>% CI 0.502, 0.673? | These data are consistent with RRs ranging fro<br>95% confiden e (ie, we are confide t that, 95 c<br>true RR will be between 0.502 and 0.673). |
| hen a clinician is aware of the | Observational bias. |
| g detects a disease earlier and thus<br>osis to death, but does not improve | Lead-time bias. |
| hic location af ects infant mortality<br>t mortality is predicted by<br>cioeconomic status is a _____. | Confounding variable. |
| have the disease and test ⊕ is the | Sensitivity. |
| ⊕s and are used to rule _____ a | Out. |
| ening test because most people<br>anergic) will have a ⊕ PPD. Highly | Highly sensitive for TB. Screening tests with hig<br>for diseases with low prevalence. |
| hic lupus erythematosus—higher | Higher prevalence. |
| higher p evalence or incidence? | Higher incidence. |
| incidence and prevalence? | Prevalence is the percentage of cases of diseas<br>one point in time. Incidence is the percentage |

| | |
|---|---|
| hetical study found an association between heart disease. How do you interpret an RR of | In patients who took ASA, the risk of h of patients who did not take ASA. |
| | In cohort studies, the odds of develop group divided by the odds of devel nonexposed group.<br>In case-control studies, the odds that by the odds that the controls were<br>In cross-sectional studies, the odds th disease divided by the odds that th disease. |
| ncer in men and the most common cause of men. | Prostate cancer is the most common causes more deaths. |
| es within 1 standard deviation (SD) of the | 68%, 95.4%, 99.7%. |
| | Number of live births per 1000 popul |
| | Number of deaths per 1000 populatio |
| e? | Number of deaths from birth to 28 da |
| | Number of deaths from birth to 1 yea (neonatal + postnatal mortality) in 1 y |
| e? | Number of deaths during pregnancy 100,000 live births in 1 year. |

| | |
|---|---|
| | failing, a given intervention has already failed, achieve the goals of care. |
| us accident. She requires emergent e not present. | Treat immediately. Consent is implied in emerg |
| ment for an STD asks that her parents n. | Minors may consent to care for STDs without p knowledge. |
| ality must be overridden. | Real threat of harm to third parties; suicidal int contagious diseases; elder and child abuse. |
| lation for medical treatment may be | When treatment noncompliance represents a s public health (eg, active TB). |
| status epilepticus, but her parents rounds. | Treat because the disease represents an imme child's life. Then seek a court order. |
| be told about her recently discovered | A physician can withhold information from the rare case of therapeutic privilege or if the patie told. |
| | |
| | |
| n onset of severe, diffuse abdominal oneal signs, and abdominal er the diaphragm. Management? | Emergent laparotomy to repair a perforated vi |
| ower GI bleeding in patients > 40 | Diverticulosis. |

| | |
|---|---|
| gers. | E coli O157:H7.<br>Bacillus cereus.<br>Salmonella.<br>Vibrio, hepatitis A virus (HAV).<br>Isospora, Cryptosporidium, Mycobacter<br>Yersinia, Campylobacter. |
| man presents with pain and watery diarrhea<br>on shows fistulas be ween the bowel and skin<br>n his tibias. | Crohn disease. |
| of the colon with an ↑ risk of colon cancer. | Ulcerative colitis (greater risk than Cro |
| stations of IBD. | Uveitis, ankylosing spondylitis, pyode<br>nodosum, 1° sclerosing cholangitis. |
| IBD. | 5-ASA agents and steroids during acu |
| h ulcerative colitis presents with fatigue, | Primary sclerosing cholangitis. |
| Mallory-Weiss and Boerhaave tears. | Mallory-Weiss—superficial ear in the<br>Boerhaave—full-thickness esophagea |
| | RUQ pain, jaundice, and fever/chills. |
| | Charcot triad plus shock and mental s |
| hepatic encephalopathy. | ↓ protein intake, lactulose, rifaximin. |
| anagement of a patient with an acute GI | Manage ABCs. |

| | |
|---|---|
| symptoms:<br>ccult blood loss, vague abdominal<br><br>in bowel movements. | Right sided: rare to have an obstruction.<br><br>Left-sided: "apple–core" lesion. |
| dehydration, muscle weakness, and | VIPoma (replace fluids and ele trolytes, may ne<br>tumor, or use octreotide). |
| der gallbladder. | Courvoisier sign (suggests pancreatic cancer). |

| | |
|---|---|
| ia. | **TICS**–**T**halassemia, **I**ron deficien y, anemia of **C**<br>**S**ideroblastic anemia. |
| mic, microcytic anemia is<br>? | Fecal occult blood test and sigmoidoscopy; sus |
| in patients with G6PD deficien y. | Sulfonamides, antimalarial drugs, fava beans. |
| use of hypercoagulability. | Factor V Leiden mutation. |
| eeding disorder. | von Willebrand disease. |
| emolytic anemia. | Hereditary spherocytosis. |
| pherocytosis. | Osmotic fragility test. |
| | Diamond-Blackfan anemia. |

| | |
|---|---|
| sents with hemarthrosis and ↑ PTT with ng time. Diagnosis? Treatment? | Hemophilia A or B; consider desmopr VIII or IX supplements. |
| ents with prolonged bleeding after dental ses, normal PT, normal or ↑ PTT, and nosis? Treatment? | von Willebrand disease; treat with des cryoprecipitate. |
| American man presents with bone pain. What ultiple myeloma reveal? | Monoclonal gammopathy, Bence Jon out" lesions on radiographs of the sku |
| | Hodgkin lymphoma. |
| sents with fever, weight loss, and night hows an anterior mediastinal mass. Suspected | Non-Hodgkin lymphoma. |
| h ↓ serum iron, ↓ total iron-binding capacity ↑ ferritin. | Anemia of chronic disease. |
| h ↓ serum iron, ↓ ferritin, and ↑ TIBC. | Iron-deficien y anemia. |
| resents with fatigue, lymphadenopathy, lated lymphocytosis. What is the suspected | Chronic lymphocytic leukemia (CLL). |
| found to have a ↓ hemoglobin and ↑ mean What are potential causes for this anemia? | ↓ $B_{12}$ (pernicious anemia, vegetarian d folate (alcoholics). |
| complication of chronic myelogenous | Blast crisis (fever, bone pain, splenom |

| | |
|---|---|
| s of fever of unknown origin (FUO). | Infection, cancer, and autoimmune disease. |
| reptococcal pharyngitis. | Fever, tender anterior cervical lymphadenopat<br>lack of cough. |
| n of streptococcal infection that is<br>nfection. | Postinfectious glomerulonephritis. |
| g factor for acute sinusitis. | Viral URI. |
| ly susceptible to these organisms. | Encapsulated organisms—pneumococcus, me<br>Haemophilus influenza , Klebsiella. |
| d on a clean-catch specimen to | $10^5$ bacteria/mL. |
| sceptible to UTIs? | Pregnant women. Treat this group aggressively<br>complications. |
| zona presents with fever, malaise,<br>nosis? Treatment? | Coccidioidomycosis; amphotericin B. |
| | 1° syphilis. |
| racteristic of what congenital | Rubella. |
| ? Treatment? | Group B strep (GBS), Ecoli, Listeria. Treat with g<br>ampicillin. |

| | |
|---|---|
| sents with soft white plaques on his tongue<br>oat. Diagnosis? Workup? Treatment? | Candidal thrush. Workup should inclu<br>nystatin oral suspension. |
| ould Pneumocystis jiroveci pneumonia<br>d in an HIV-⊕patient? Mycobacterium avium–<br>laxis? | ≤ 200 for P jiroveci (with TMP-SMX); ≤ 5<br>clarithromycin/azithromycin). |
| ephritis. | Pregnancy, vesicoureteral reflux, an t<br>catheters, kidney stones. |
| stchemotherapy. | 7–10 days. |
| | Lesion of 1° Lyme disease. |
| gs or endocarditis. | Fever, heart murmur, Osler nodes, spli<br>lesions, Roth spots. |
| cell disease. | Parvovirus B19. |

| | |
|---|---|
| ral infection | Actinomyces israelii |
| rtially acid-fast in lung infection | Nocardia asteroides |
| | Haemophilus ducreyi |
| | Pasteurella multocida |
| | Sporothrix schenckii |
| tal muscle cysts | Trichinella spiralis |
| iver cysts | Echinococcus granulosus |
| | Enterobius vermicularis |
| ith pets | Toxoplasma gondii |
| | Neisseria meningitidis |

| | |
|---|---|
| uspected? | Staureus or Staphylococcus epidermidis. |
| s in a native valve after having a | Streptococcus viridans. |
| | |
| y standing and walking and relieved f the hips. | Spinal stenosis. |
| heumatoid arthritis. | MCP and PIP joints; DIP joints are spared. |
| rsen over the course of the day and | Osteoarthritis. |
| iated with multiple fractures and mistaken for child abuse. | Osteogenesis imperfecta. |
| tiffness th t improves with activity worsens at rest. Diagnostic test? | Suspect ankylosing spondylitis. Check HLA-B2 |
| thritis in young men. Associated | Reactive (Reiter) arthritis. Most commonly asso Also consider Campylobacter, Shigella, Salmone |
| excruciating first TP joint pain ne. Diagnosis, workup, and chronic | Gout. Needle-shaped, negatively birefringent c joint fluid aspi ate. Chronic treatment with allo |
| irefringent crystals on joint fluid | Pseudogout. |

| | |
|---|---|
| t for migraine. | Antihypertensives, antidepressants, a |
| uitary tumor. Treatment? | Prolactinoma. Dopamine agonists (eg |
| presents with acute "broken speech." What<br>lobe and vascular distribution? | Broca aphasia. Frontal lobe, left MCA c |
| use of subarachnoid hemorrhage (SAH). | Trauma; the second most common is |
| perdensity on CT that does not cross the | Subdural hematoma—bridging veins |
| r initial altered mental status with an<br>rval. Diagnosis? Most likely source? Treatment? | Epidural hematoma. Middle meninge<br>evacuation. |
| | ↑ ICP, RBCs, xanthochromia. |
| ssociation. | Guillain-Barré syndrome (↑ protein in<br>cell count). |
| to a patient's ear, and the fast phase of the<br>he opposite side. Normal or pathologic? | Normal. |
| sources of metastases to the brain. | Lung, breast, skin (melanoma), kidney |
| n who are accused of inattention in class and<br>th ADHD. | Absence seizures. |
| esentation of intracranial neoplasm. | Headache. 1° neoplasms are much les<br>metastases. |
| use of seizures in children (2–10 years). | Infection, febrile seizures, trauma, idio |

| | |
|---|---|
| ress to choreiform movements, d altered behavior. | Huntington disease. |
| a port-wine stain in the $V_1$ llectual disability, seizures, and oma. | Sturge-Weber syndrome. Treat symptomatically cerebral resection of the af ected lobe. |
| kin. | Neurofib omatosis type 1. |
| yperorality, and hyperdocility. | Klüver-Bucy syndrome (amygdala). |
| otomatic patient to diagnose | Edrophonium. |

| | |
|---|---|
| eding. | Placental abruption and placenta previa. |
| oss appearance of complete | Snowstorm on ultrasonography. "Cluster-of-gra gross examination. |
| plete mole. | 46,XX. |
| tal tissue. | Partial mole. |
| on. | Continuous, painful vaginal bleeding. |
| | Self-limited, painless vaginal bleeding. |
| e performed with suspected placenta | Never. |

| | |
|---|---|
| m when a woman presents with amenorrhea. | β-hCG; the most common cause of an |
| ng during and between menstrual periods. | Menometrorrhagia. |
| with normal prolactin, no response to e challenge, and a history of D&C. | Asherman syndrome. |
| ovarian syndrome. | Weight loss and OCPs. Consider metfo |
| duce ovulation. | Clomiphene citrate. |
| ed in a postmenopausal woman who presents | Endometrial biopsy. |
| l treatment of ectopic pregnancy. | Patient stable; unruptured ectopic pre weeks' gestation. |
| ndometriosis. | OCPs, danazol, GnRH agonists. |
| in endomet iosis. | Powder burns, "chocolate cysts." |
| cation for an ectopic pregnancy. | Ampulla of the oviduct. |
| follow a leiomyoma. | Ultrasonography. |
| omyoma. | Regresses after menopause. |
| l discharge and petechial patches in the vix. | Trichomonal vaginitis. |

| | |
|---|---|
| nale infertility. | Endometriosis. |
| ypical squamous cells of CUS) on Pap smear. Follow-up | Colposcopy and endocervical curettage. |
| uture risk of invasive carcinoma in | Lobular carcinoma in situ. |
| | |
| sociated with ↑ vanillylmandelic acid HVA). | Neuroblastoma. |
| heoesophageal fistula TEF). | Esophageal atresia with distal TEF (85%). Unab |
| ation. | Mild illness and/or low-grade fever, current ant prematurity. |
| rauma. | Ophthalmologic exam, CT, and MRI. |
| | Cystic fib osis (Hirschsprung disease is associat meconium for 48 hours). |
| er the first eeding. | Duodenal atresia. |
| with nonbilious projectile emesis. priate steps in management? | Pyloric stenosis. Hydrate and correct metabolic correct pyloric stenosis with pyloromyotomy. |

| | |
|---|---|
| ancer? | |
| onth well-child visit. | HBV, DTaP, Hib, IPV, PCV, rotavirus. |
| ear-old girl. | Precocious puberty. |
| ays with epidemics in winter and spring. | RSV bronchiolitis. |
| S. | Surfactant deficien y. |
| ols, colicky abdominal pain, bilious vomiting, mass in the RUQ. | Intussusception. |
| ease that causes 2° hypertension. What would xamination? | Coarctation of the aorta; ↓ femoral pu |
| r otitis media. | Amoxicillin. |
| thogen causing croup. | Parainfluenza virus ype 1. |
| all for his age and has peeling skin and a | Kwashiorkor (protein malnutrition). |
| syndrome with mental retardation, gout, self- oathetosis. | Lesch-Nyhan syndrome (purine salvag deficien y). |
| ontinuous "machinery murmur." What drug | Patent ductus arteriosus (PDA). Indom PDA. |
| posterior neck mass and swelling of the | Turner syndrome. |

| | |
|---|---|
| r husband yells at her child.<br>best friend acts overly kind.<br>sly murder.<br>egins to wet his bed. | Displacement.<br>Reaction formation.<br>Isolation.<br>Regression. |
| y, high fever, autonomic instability,<br>e phosphokinase. | Neuroleptic malignant syndrome. |
| (< 85%), bradycardia, and abnormal<br>. | Anorexia. |
| nt episodes of palpitations,<br>ding doom.. | Panic disorder. |
| clozapine. | Agranulocytosis. |
| ns of social withdrawal, worsening<br>ncrete thinking. | Schizophreniform disorder (a diagnosis of schi<br>months of symptoms). |
| psychotics. | Weight gain, type 2 DM, QT-segment prolonga |
| Vhaloperidol and complains that his<br>agnosis? Treatment? | Acute dystonia (oculogyric crisis). Treat with be<br>diphenhydramine. |
| with a history of alcohol withdrawal | Neuroleptics, which can lower the seizure thre |
| of theft, vandalism, and violence | Conduct disorder. Associated with antisocial p<br>adults. |

| | |
|---|---|
| used as a child frequently feels outside of or dy. | Depersonalization disorder. |
| nt takes haloperidol for 1 year and develops e movements. Diagnosis? Treatment? | Tardive dyskinesia. ↓ or discontinue h antipsychotic (eg, risperidone, clozap |
| ressive disorder is counseled to avoid ith his new medication. What class of ng? | MAOIs. |
| | |
| | Stasis, endothelial injury, and hyperco |
| effusion | Pleural/serum protein > 0.5; pleural/s |
| ffusion | Think of leaky capillaries. Malignancy, pulmonary embolism (PE) with infarc |
| e effusion | Think of intact capillaries. CHF, liver or losing enteropathy. |
| patient having an asthma exacerbation may | Fatigue and impending respiratory fa |
| sthma and COPD exacerbations. | $\beta_2$-agonists and corticosteroids (antic COPD exacerbation as well). |
| | Dyspnea, lateral hilar lymphadenopat noncaseating granulomas, ↑ angioter |

| | |
|---|---|
| ndrome of inappropriate antidiuretic | Small cell lung cancer (SCLC). |
| mbert Eaton syndrome. | SCLC. |
| cigarette exposure. | SCLC, SCC. |
| with acute shortness of breath. | Spontaneous pneumothorax. Spontaneous reg $O_2$ may be helpful. |
| horax. | Immediate needle thoracostomy (over diagnos |
| oma in an isolated pulmonary nodule. | Age > 45–50; tobacco use; lesions new or large films; absen e of calcific tion or irregular calcif irregular margins. |
| | Hypoxemia and pulmonary edema with norma wedge pressure (PCWP). |
| . | Pulmonary fib osis, pleural plaques, bronchog lung field), mesothelioma (pleu al mass). |
| icosis? | Mycobacterium tuberculosis. |
| | Right-to-left shunt, hypoventilation, low inspir defect, V/Q mismatch. |
| ngs or pulmonary edema. | Cardiomegaly, prominent pulmonary vessels, I "bat's-wing" appearance of hilar shadows, and peribronchial cuffi . |
| ggesti e of PE. | Westermark sign and Hampton hump. |

| | |
|---|---|
| idened QRS. | Hyperkalemia. |
| emia. | C BIG K (calcium gluconate, bicarb, ins |
| r moderate hypercalcemia. | IV hydration. |
| t with $Fe_{Na}$ < 1%. | Prerenal. |
| sents with acute-onset flank pain and | Nephrolithiasis. |
| pe of nephrolithiasis. | Calcium oxalate. |
| nrolithiasis. | Noncontrast CT. |
| vs bilateral enlarged kidneys with cysts. naly? | Cerebral berry aneurysms (autosomal disease, PCKD). |
| ion, and oliguria. | Nephritic syndrome. |
| minemia, hyperlipidemia, hyperlipiduria, and | Nephrotic syndrome. |
| rm of nephrotic syndrome in adults. | Focal segmental glomerulosclerosis. |
| resenting 3 days after URI, **normal C3.** | IgA nephropathy (Berger disease). |
| ralgias, abdominal pain. | Henoch-Schönlein purpura. |
| ith deafness. | Alport syndrome. |
| ith hemoptysis. | Granulomatosis with polyangiitis and |

| | |
|---|---|
| f bladder cancer. | Transitional cell carcinoma. |
| rrection of hyponatremia. | Central pontine myelinolysis. |
| hat type of acid-base disorder? | Anion gap acidosis and 1° respiratory alkalosis respiratory stimulation. |
| nly seen in pregnant women. | Respiratory alkalosis. |
| h irritative and obstructive urinary | Probably BPH. Options include no treatment, t surgical intervention (TURP). |

## RGENCY MEDICINE

| | |
|---|---|
| yndrome of muscle rigidity, bility, and extrapyramidal symptoms. | Antipsychotics (neuroleptic malignant syndron |
| | Acute mania, immunosuppression, thin skin, o bruising, myopathies. |
| | Benzodiazepines. |
| overdose. | N-acetylcysteine. |
| | Naloxone. |
| overdose. | Flumazenil (monitor for withdrawal and seizure |
| gnant syndrome and malignant | Dantrolene. |

| | |
|---|---|
| e of aortic disruption or dissection. | Widened mediastinum (> 8 cm), loss c tracheal deviation to the right, depres |
| ons for surgery in patients with acute | Free air under the diaphragm, extrava bowel distention, space-occupying le (angiography). |
| ganism in burn-related infections. | Pseudomonas. |
| fluid epletion in burn patients. | Parkland formula: 24-hour fluids = 4 × |
| ut in a trauma patient. | 50 cc/hr. |
| ut in a stable patient. | 30 cc/hr. |
| hock. | Hypotension and bradycardia. |
| g triad). | Hypertension, bradycardia, and abnor |
| heral vascular resistance (PVR). | Hypovolemic shock. |
| | Cardiogenic (or obstructive) shock. |
| | Distributive (eg, septic or anaphylacti |
| ock. | Fluids and antibiotics. |
| nic shock. | Identify cause; inotropes (eg, dopami |
| emic shock. | Identify cause; fluid and blood epleti |
| actic shock. | Epinephrine 1:1000 and diphenhydra |

eterioration. Convex shape on CT.

ase 544 OB/GYN
nterest Statement 545 Pediatrics
546 Psychiatry
546 Surgery
ergency Medicine, Family Medicine 547 Commercial Review Courses
547

disciplines (internal medicine, neurology, OB/GYN, pe
and surgery). Within each section, resources are arranged
by Author, and finally by Title.

For this ninth edition of First Aid for the USMLE Step 2
review resources has been completely revised, with in-de
ments on more than 100 books and online and mobile a
rating scale with six different grades reflects the detailed
Each resource receives a rating as follows:

| | |
|---|---|
| A+ | Excellent for boards review. |
| A<br>A– | Very good for boards review; choose among the gr |
| B+<br>B | Good, but use only after exhausting better sources |
| B– | Fair, but there are many better books in the discip<br>subject material. |

The **Rating** is meant to reflect the overall usefulness of
paring for the USMLE Step 2 CK exam. This is based on
including:

- The cost of the resource
- The readability of the text
- The appropriateness and accuracy of the resource
- The quality and number of sample questions
- The quality of written answers to sample questions
- The quality and appropriateness of the illustrations (e
photographs)

d books not included in our database, for evaluation. We of new books or suggestions for alternate modes of study n preparing for the examination, such as flash cards, com-, commercial review courses, and Internet Web sites.

## LICT-OF-INTEREST STATEMENT

ook, including the ratings, reflects the opinion or influence errors and omissions will gladly be corrected if brought to authors through our blog at www.firstaidteam.com. Please x and the entire First Aid for the USMLE series are publi- authors of this book; their ratings are based solely on rec- the student authors of this book as well as data from the eedback forms.

| | | | |
|---|---|---|---|
| | | McGraw-Hill Education, 2010, 2nd ed., 741 pages, ISBN 9780071625715 | Review/ |
| | Van Kleunen | Lippincott Williams &Wilkins, 2014, 3rd ed., 352 pages, ISBN 9781451189599 | Review |
| E | Ayala | Lippincott Williams &Wilkins, 2012, 5th ed., 608 pages, ISBN 9781451144062 | Review |
| | Carl | Medmaster Books, 2012, 5th ed., 402 pages, ISBN 9780940780996 | Review |
| | Fischer | Kaplan Publishing, 2012, 3rd ed., 408 cards, ISBN 9781609780371 | Flashcard |
| | Ibsen | Lippincott Williams &Wilkins, 2012, 4th ed., 384 pages, ISBN 9780781787390 | Review/T |
| | McGraw-Hill | McGraw-Hill Education, 2009, 5th ed., 292 pages, ISBN 9780071604635 | Test/368 |
| t | Bernstein | McGraw-Hill Education, 2011, 7th ed., 442 pages, ISBN 9780071633017 | Test/500 |
| | Kaplan Medical | Kaplan Publishing, 2014, 6th ed., 540 pages, ISBN 9781419550485 | Test/850 |
| | McWilliams | Kaplan Publishing, 2014, 2nd ed., 480 pages, ISBN 9781609788988 | Review |
| | Naheedy | McGraw-Hill Education, 2010, 2nd ed., 348 pages, ISBN 9780071627160 | Review |
| | O'Connell | Elsevier, 2013, 4th ed., 352 pages, ISBN 9781455703111 | Review |
| | Ryan | Lippincott Williams &Wilkins, 2010, 2nd ed., 385 pages, ISBN 9781605479071 | Review |

| | | |
|---|---|---|
| sment & | Rosh | McGraw-Hill Education, 2012, 3rd ed., 624 pages, ISBN 9780071773102 |
| mergency<br>lp | Stead | McGraw-Hill Education, 2011, 3rd ed., 537 pages, ISBN 9780071739061 |
| Medicine | Toy | McGraw-Hill Education, 2012, 3rd ed., 625 pages, ISBN 9780071753951 |
| al | Toy | McGraw-Hill Education, 2013, 4th ed., 560 pages, ISBN 9780071761727 |
| | Harward | Elsevier, 2012, 5th ed., 624 pages, ISBN 9780323063982 |
| PreTest<br>&Review | Knutson | McGraw-Hill Education, 2012, 3rd ed., 303 pages, ISBN 9780071760522 |
| | Bergin | Lippincott Williams &Wilkins, 2011, 4th ed., 848 pages, ISBN 9781605476759 |
| ncy | Caterino | Lippincott Williams &Wilkins, 2003, 1st ed., 316 pages, ISBN 9781405103572 |
| ily | Perez | McGraw-Hill Education, 2011, 2nd ed., 402 pages, ISBN 9780071715157 |
| rnal | Saadat | McGraw-Hill Education, 2011, 2nd ed., 254 pages, ISBN 9780071715171 |
| Self-<br>view | Smalligan | McGraw-Hill Education, 2012, 13th ed., 470 pages, ISBN 9780071761499 |
| ne | Young | Lippincott Williams &Wilkins, 2009, 5th ed., 448 pages, ISBN 9780781788700 |
| rgency | Jang | McGraw-Hill Education, 2011, 2nd ed., 480 pages, ISBN 9780071715188 |

| | | | |
|---|---|---|---|
| y. | Schneider | McGraw-Hill Education, 2012, 15th ed., 592 pages, ISBN 9780071761277 | Test/500 |
| | Miller | McGraw-Hill Education, 2011, 2nd ed., 490 pages, ISBN 9780071715133 | Review |
| | Pfeifer | Lippincott Williams &Wilkins, 2011, 7th ed., 528 pages, ISBN 9781608315765 | Review/T |
| d | Katz | McGraw-Hill Education, 2011, 9th ed., 404 pages, ISBN 9780071712132 | Test/130 |

| AUTHOR | PUBLISHER | TYPE |
|---|---|---|
| Stead | McGraw-Hill Education, 2011, 3rd ed., 607 pages, ISBN 9780071664035 | Review |
| Toy | McGraw-Hill Education, 2013, 4th ed., 479 pages, ISBN 9780071766982 | Review |
| Yetman | McGraw-Hill Education, 2013, 13th ed., 422 pages, ISBN 9780071761239 | Test/500 |
| Davey | McGraw-Hill Education, 2011, 2nd ed., 307 pages, ISBN 9780071715140 | Review |
| Marino | Lippincott Williams &Wilkins, 2013, 6th ed., 416 pages, ISBN 9781451116045 | Review/T |
| Polin | Elsevier, 2011, 5th ed., 640 pages, ISBN 9780323065610 | Review |
| Jackson | McGraw-Hill Education, 2010, 7th ed., 326 pages, ISBN 9780071475686 | Test/100 |
| McGahren | Lippincott Williams &Wilkins, 2011, 4th ed., 542 pages, ISBN 9781605476766 | Review |

| | AUTHOR | PUBLISHER |
|---|---|---|
| gical gnettes Vards | Pestana | Kaplan Publishing, 2013, 1st ed., 240 pages, ISBN 9781609789169 |
| y | Toy | McGraw-Hill Education, 2012, 4th ed., 522 pages, ISBN 0071766995 |
| urgery | Kaufman | McGraw-Hill Education, 2009, 2nd ed., 537 pages, ISBN 9780071448710 |
| ry | Zaslau | Lippincott Williams &Wilkins, 2014, 2nd ed., 408 pages, ISBN 9781451187632 |
| | Blackbourne | Lippincott Williams &Wilkins, 2014, 7th ed., 824 pages, ISBN 9781451192919 |
| | Jarrell | Lippincott Williams &Wilkins, 2008, 5th ed., 647 pages, ISBN 9780781759014 |
| Self- view | Kao | McGraw-Hill Education, 2012, 13th ed., 374 pages, ISBN 9780071761215 |
| gery | Tevar | McGraw-Hill Education, 2012, 2nd ed., 412 pages, ISBN 0071715126 |
| ical | Harken | Elsevier, 2009, 6th ed., 517 pages, ISBN 9780323057110 |
| gery | Karp | Lippincott Williams &Wilkins, 2008, 5th ed., 253 pages, ISBN 9780781788687 |

he review courses you are considering. In addition, course conten
ent opinions can be found in discussion groups on the World Wide W
he Step 2 CK commercial review courses.

**ation**

e.

800-527-8378)
lan.com

ooltutors.com
s.com

**view**

sternmedicalreview.com
edicalreview.com

eekly circulation of 9000. Tao earned his medical degree from
completed his residency training in internal medicine at Yale
wship training at Johns Hopkins University. Tao subsequently
Medsn, a medical education technology venture, and served as
ficer. He is currently conducting research in asthma education
Louisville.

## MD

itor, entrepreneur, and teleradiologist on sabbatical. In 1990 he
ored the original First Aid for the USMLE Step 1. His entrepre-
nclude a student-focused medical publisher (S2S), an e-learning
ol.com/Medsn), and an ER teleradiology practice (24/7 Radiol-
ed to the Left Coast, Vikas completed a bachelor's degree at the
rnia, Berkeley; an MD with thesis at UCSF; and a diagnostic ra-
UCLA. His eclectic interests include technology, information
, South Asian diasporic culture, and avoiding a day job. Always
rtcut, Vikas is an adventurer, knowledge seeker, and occasional
s novice status as a kiteboarder and single father, and strives to
global citizens.

## MD

a resident in internal medicine at Stanford University Medical
hospital, he enjoys photography, reading and watching Shake-
exploring the Bay Area's many attractions. Vincent completed
ee in chemistry at Amherst College and his medical degree at
chool, where he graduated magna cum laude. Between college
, he spent a year as a Fulbright fellow in Frankfurt, Germany.
nultiple basic science research projects, most recently in intesti-
well as clinical research in liver cancer. He plans to specialize




d USMLE-style questions with
tions

e interface for true simulation

test mode, subject matter, and